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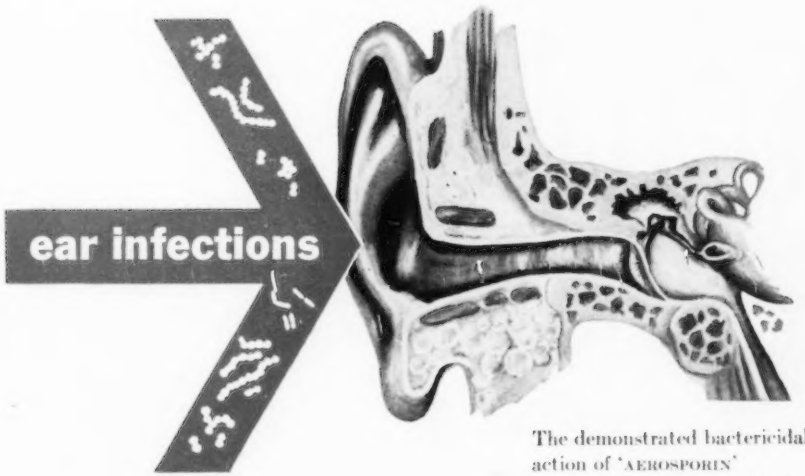

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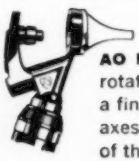

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Reporting on

PROGRESS IN OPHTHALMOLOGY AND OTOLARYNGOLOGY

Volume I 1952

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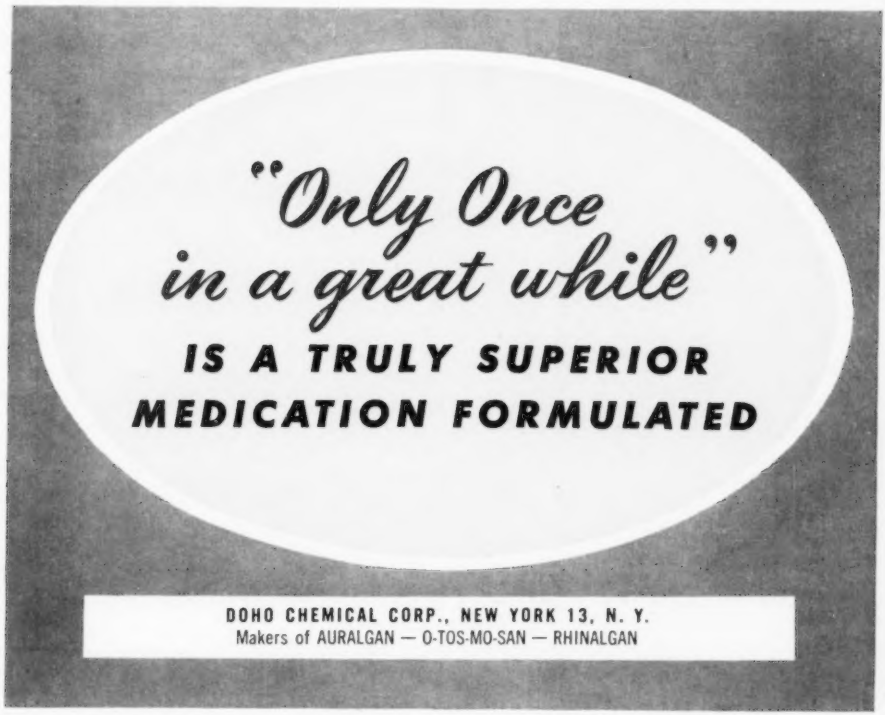
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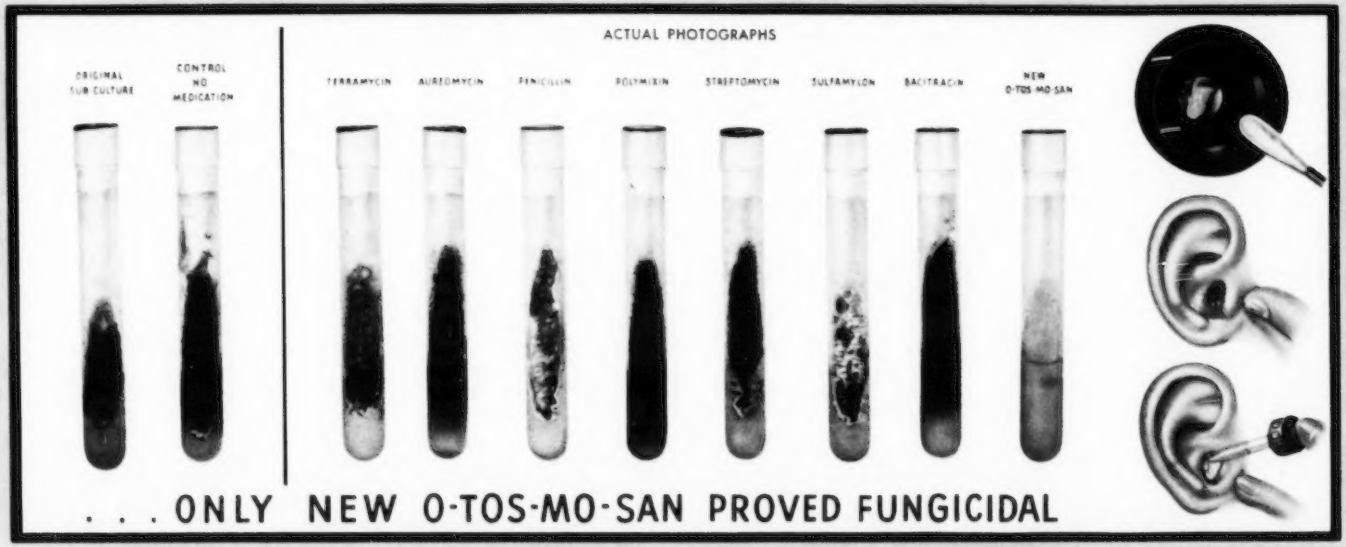

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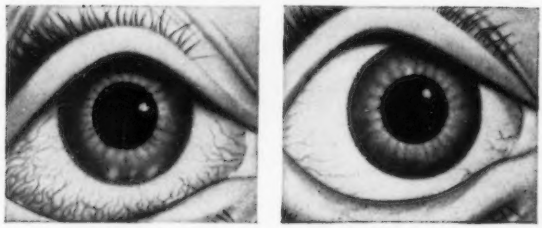

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1. Scheie, H. G., Tyner, G. S., Buesseler, J. A., and Alfano, J. E., *J. A. M. A. Arch. Ophth.* 45:301, March 1951.
2. Leopold, I. H., Purnell, J. E., Cannon, E. J., Steinmetz, C. G., and McDonald, P. R., *Am. J. Ophth.* 34:361, March 1951.

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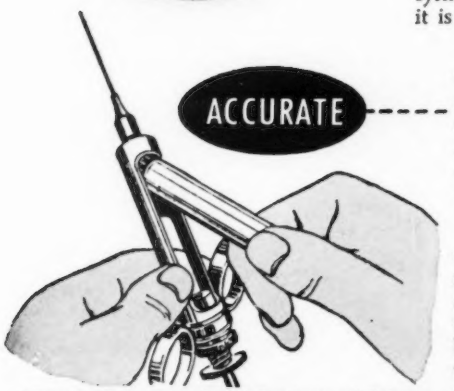

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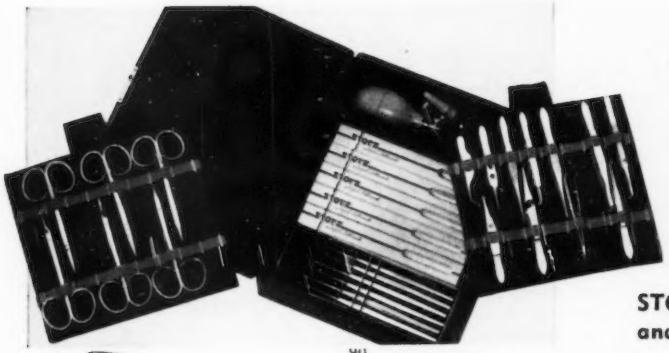

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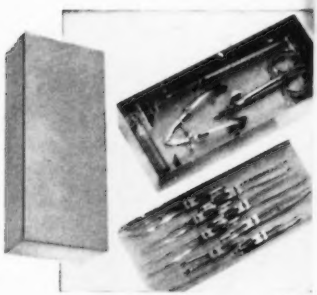

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BY INVITATION

THOUGH many years have passed since Gonin invented the surgical treatment of retinal detachment and although the operative technic of this is not difficult, yet this technic is by no means widespread and there are in all countries a number of colleagues that avoid this operation, although they are expert surgeons who include all other ocular operations in their field. This situation is due to several factors.

The first of these is the diversity of clinical types of this disease which necessitates a different technic for each of them. After visiting several ophthalmic centers to watch operations, an oculist will acquire a standard procedure for such operations as cataract, glaucoma, squint and so forth. In detachment, however, he finds considerable variations in technic and, therefore, does not develop a standard plan of procedure. The reason for this is that the operative technic of retinal detachment cannot be standardized. Another reason is the relatively small number of these operations, which means that oculists, unless they are in a large city or a large hospital, do not have the opportunity of seeing many of them. This limits their experience in this branch of surgery.

The higher incidence of myopia in Europe, especially in the Latin countries, than in North American means that the condition is commoner there.

Presented at the Fifty-Sixth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 14-19, 1951, Chicago, Ill.

Further, another factor of great importance to be found is the difficulty of examining the fundus where the media are opaque. This is of great importance in the United States where few surgeons use indirect ophthalmoscopy. In order that the ophthalmoscopic examination should be complete and efficient, it is necessary that both methods, direct and indirect, but especially the latter, be employed.

As Gonin said, "The instrument that cures retinal detachment is the retinoscope." His pupils propagated this truth, but it is difficult for many colleagues to accustom themselves to long and repeated examinations of the fundus. There are, indeed, cases which test the oculist's patience, but the work in the dark room is far more important than that which is done in the operating theater, as the efficiency of the latter depends upon the perfection of the former.

These concepts are well known but do not have the influence that they should. Those who have a large experience with retinal detachment will confirm that many failures are due either to defective localization of the tears or to an excess of diathermic action. The latter follows from the first, that is to say, where tears are badly localized a more extensive diathermic application is made. This is easy for the surgeon and hard on the patient, as the larger the surface of choroid coagulated, the great-

er the local reaction and the smaller the surface of healthy choroid remaining for reabsorption of the subretinal fluid. However considerable the drainage, a certain amount of this always remains after operation.

In view of the fact that many ophthalmologists have only a limited experience with this condition, it remains for those of us who have had a larger experience to hand on the results of this experience that we believe might be useful to them.

In this paper I will attempt to classify the different types of retinal detachment in order to systematize as far as possible the necessary procedure.

Finally, I will give the approximate percentage of cures obtained, though this depends on many factors, such as the size of the tears, the turbidity of the media, the age of the patient, the duration of the disease and, mainly, the improvement with rest. I shall mention two percentages of cure for each type. The first refers to those cases which did not improve with binocular bandage and rest in bed, the second to those which did improve with rest.

In many of the described types of retinal detachment, the tears may be invisible either owing to their small size or because they are hidden by folds or vitreous turbidity. There is also the possibility that they do not exist, the detachment being due to choroidal exudation. However, tears may exist in cases of choroidal exudation. The mechanism in such cases is as follows: At one point in old chorioretinitis the retina adheres to the choroid, the exudation elevates the retina and tears it, and a small piece of retina remains adhered to the choroid. It is probable that tears of the macula are produced by this mechanism. The tear is circular, but here the operculum is not seen floating in the vitreous as happens in circular tears of other zones of the retina where the operculum is adherent to the framework of the vitreous.

Trauma has not as much importance as was formerly believed in the etiology of the detachment, as the retina tears if it is degenerated or atrophied. It is therefore convenient, even in cases where the traumatic factor exists, to eliminate other possible causes, especially focal sepsis, as the two main causes of retinal detachment, myopic and senile degeneration, are not treatable.

Detachments of the types 1, 5, 9 and 10, if not operated upon, are displaced downwards, because the subretinal fluid, its density increasing with time, descends to the inferior pole and the separation of the superior retina becomes smaller. If the tear is large (type 6, 8, 11, 13) and one does not operate, the detachment extends in all directions, as a rule, but not downwards with reapplication of the superior retina, because here the density of the subretinal fluid does not increase much, as there is a wide communication with the vitreous.

When there is a small amount of subretinal fluid, pressure upon the globe is not necessary to expel it, nor is the injection of air into the vitreous. It might even happen that quite a large amount of subretinal fluid remains unexpelled and that a cure is attained. Cases are seen frequently with big retinal bulges which reabsorb with preoperative rest. Nevertheless, if there is a moderate amount of subretinal fluid, it is preferable to expel it.

The detachments which reach the oculist latest are the inferior ones, as they are the slowest to reach the macular region and the scotoma they produce is in the least sensitive part of the retina corresponding to the superior visual field hidden by the eyebrow. The detachments of the nasal zone are also slow to alarm the patient as they, too, reach the macula late.

SYNTHESIS OF THE BASIC CONCEPTS OF THE EVOLUTION AND TREATMENT OF RETINAL DETACHMENT

Whenever hemorrhagic opacities are present in the vitreous one must consider the possibility that they may be followed by retinal detachment.

When a detachment with a small tear increases in spite of rest, it is wiser not to operate, as the choroid may react with the subretinal fluid and, instead of absorbing it, exude more.

Rest implies *binocular bandage* as a rest for the oculomotor muscles. This is more efficacious than rest for the body, though the latter, too, has value.

When nonperforating diathermy is applied at the beginning of the operation in order to obtain a white spot of coagulated retina for orientation, it is convenient, if there is subretinal fluid, to push the electrode against the sclera to depress it, as a white spot of coagulation would not be obtained without depressing the electrode. If the separation of the retina from the choroid is very extensive, the coagulation of the former is not attained.

Once the sclera and the choroid have been perforated to allow the subretinal fluid to exude, a certain amount of it does so, but if the globe is pressed or air is injected into the vitreous, it will be frequently observed that a further large quantity (which often astonishes the operator) follows it.

When the globe is pressed to obtain issue of subretinal fluid, one can see vitreous going out when puncture has been done exactly at the place of the tear. This is not a bad sign but, on the contrary, a good one, as adherence of the vitreous framework to the cicatrix on the wall of the globe contributes to maintenance of the retina at this point.

The injection of air is generally performed near the insertion of a rectus muscle, whose tendon is held by forceps in order to prevent the needle depressing the wall of the globe too much. It is convenient to make the puncture at about 6 or 7 millimeters from the limbus in the direction of the posterior pole and to inject a quantity of air sufficient to evacuate the subretinal fluid.

When diathermy has been applied to the zone of sclera corresponding to the tears, it is prudent to surround the intensely diathermized zone by a very slight diathermic action. This is to avoid relapses due to tractions in the border of the intensely diathermized zone, as slight diathermy produces a zone of retinochoroidal adhesion without the destruction of these membranes, which occurs when they are much coagulated.

Postoperative rest with both eyes bandaged takes eight days for the most favorable cases and perhaps three or four weeks for severe ones. The most convenient position according to the situation of the tears is still a matter of discussion. The really important thing is that the immobilization must be as perfect as possible. The position of the head which would appear the most logical is that of rest on the side where the evacuating puncture has been done. In cases of superolateral or lateral tears with bulges in the inferior pole, if the puncture has been done in the inferior pole, as many of my colleagues do, the head should not be resting on the side where the tears were, because of the subretinal fluid which may remain after the operation and which, being more dense than the vitreous, will move to the zone of the tears and separate the retina from the choroid. If the evacuating puncture was made in the zone of the tears, then the patient's head may rest on the side where the tears were.

In severe cases and in cases of relapse during the first months after the operation, diathermic puncture followed by air injection may bring about a cure.

And here I conclude this paper, though I am afraid it contains nothing

new for those who are experienced in retinal detachment, I will be very glad if it has at least been of some interest for those who are not very familiar with this branch of ophthalmology.

LEGENDS FOR COLOR PLATES

FIG. 1—Incipient superoexternal detachment. This is the most frequent type, as 30 per cent of all cases begin in this way. In the stage shown in the figure, the patient rarely consults the oculist. He does not become alarmed until the condition is more advanced, as he notices only a slight reduction of the visual field in the nasal region and a few photopsias.

Treatment. If the retinal prominence is not reduced, diathermy and puncture. If the prominence is reduced, diathermy without puncture. Cure, 85-98 per cent.

FIG. 2—Case 1 after some days. In this stage we see most of these patients with this type of detachment, as it reaches the macular region and thus alarms the patient by the important loss of central vision.

Treatment. Rest in order to obtain as large a reapplication of the retina as possible, diathermy operation with puncture and expulsion of the subretinal fluid. Cure 80-95 per cent.

FIG. 3—Cases 1 and 2, further advanced. This is the appearance which figures 1 and 2 take on if the patient neither rests nor has an operation.

Treatment. Rest until, if possible, the eye recovers the appearance of figure 2 or 1. At this point, operative intervention. The operation should consist of diathermy and puncture, which should be done in the region of the tear, emptying the subretinal fluid by pressure or air injection. Cure, 50-80 per cent.

FIG. 4—Total detachment. This is a further advanced case of figures 1, 2 and 3.

Treatment. Rest. If the detachment came on rapidly in spite of a small tear and is not reduced by rest, the prognosis is grave, as an exaggerated choroidal reaction is thus suggested. Rest must be continued and a search made for cause, mainly focal sepsis. If the detachment became total relatively slowly, i.e., within about two to three months, the prognosis is not so bad. Reapplication may be obtained with rest, thus allowing an operation, although macular vision will remain diminished.

Treatment. Diathermy in the region of the tear and puncture with expulsion of subretinal fluid. Cure, 20-60 per cent.

FIG. 5—Detachment with superointernal or superonasal tear. This type is always less serious than those occurring on the outer side, as the optic nerve preserves the macula.

Treatment. Rest, lying on the side of the detachment in order to avoid movement of the subretinal fluid to the temporal side. Operation is employed when the retina has reached its maximum reapplication.

Treatment. Application of diathermy and expulsion of subretinal fluid. Cure, 60-95 per cent.

FIG. 6—Recent superior detachment with a big tear. When the tear in the superior hemisphere is large, the detachment rapidly becomes prominent and extensive, as the vitreous has a large opening through which to pass into the subretinal space. Reapplication with rest is, therefore, much more difficult.

Treatment. An attempt should always be made to improve the position of the retina with rest, but if there is no improvement after a few days, operate. It is useful in such cases, quite apart from the diathermy application in the region of the tear, to make a puncture in this zone and press on the eyeball so that

some vitreous is evacuated after the subretinal fluid. The fixation of the vitreous structure at the exterior of the globe favors reapplication of the retina. Cure, 50-90 per cent.

FIG. 7A — Recent superior detachment of ballooned type without visible tear. Though this may be a detachment due to choroidal exudation, the probability is that the tear is hidden by the prominent retina.

FIG. 7B — In this case one must not forget that the zone of the sclera which corresponds to the point where the tear is localized may be more anterior than that which appears in the ophthalmoscopic examination prior to the evacuation of subretinal fluid.

Treatment. Rest. Sometimes the tear then becomes visible with rest, as in figure 7B. If the tear remains invisible after rest, one must operate, employing a small puncture, a procedure which almost invariably makes the tear visible. In this way the diathermy can be placed in the proper position towards the end of the operation. Retinal fluid is expelled. Cure, 75-80 per cent.

FIG. 8—Recent superior detachment with several tears. This is a case similar to figure 6, but of greater gravity for two reasons: first, because the multiple perforations indicate great fragility of the retina and, second, because the operation is more laborious and obliges one to make an extensive diathermic application, thus diminishing the probabilities of cure.

Treatment. Diathermy at the zone of the tears and punctures with expulsion of the subretinal fluid. Cure, 30-70 per cent.

FIG. 9—Detachment of a few weeks standing with small tears in a zone of reticulovacuolar degeneration in the superoexternal region. In spite of a long and narrow degenerated zone, the prognosis is benign, as the said zone is usually confined to a superoexternal sector in the equatorial region. The tears being very small, the detachment usually progresses very slowly. A similar degenerative lesion is often present in a symmetrical position in the fellow eye.

Treatment. Nonperforating diathermy and puncture if there was subretinal fluid after the rest. Cure, 70-90 per cent.

FIG. 10—Detachment of some months standing with tear above and detachment below. When the tear is small the detachment grows slowly. When after some weeks the density of the subretinal fluid increases, the fluid descends to the inferior hemisphere of the eye and the retina in the region of the tear approaches the choroid.

Treatment. Diathermy at the zone of the tear with puncture in the same region and pressure or air injection in order to evacuate subretinal fluid. Cure, 50-80 per cent.

FIG. 11—Extensive detachment with an enormous tear. Rest is useless in most of these cases because of the large communication between the vitreous and the subretinal space.

Treatment. After rest, diathermy is applied in the region of the tear. Puncture and pressure upon the globe, or air injection; protrusion of a vitreous bead through the puncture is considered of favorable import. Cure, 5-30 per cent.

FIG. 12—Multiple tears with an almost total detachment. A very severe type indicating a degenerative condition of the retina or multiple adherences of the vitreous frame to the retina.

Treatment. Extensive diathermy after some days of rest. Puncture. Expulsion of the subretinal fluid. Cure, 5-30 per cent.

FIG. 13—Retinal detachment due to superior disinsertion. A rare case and frequently due to traumatism. Little improvement is obtained with rest, a finding common to all cases of disinsertion of the ora serrata.

Treatment. Diathermic application, forming a line limiting the zone of the tear by a barrier of adhesive chorioretinitis. Several diathermal punctures in the diathermized zone. It is convenient to press the globe so that the normal vitreous issues through these punctures. It is impossible to reapply the retina of the tear to its original position. Cure, 40 per cent.

FIG. 14—Extensive detachment with tear at the equator and at the macula. In this case the detachment is initially superior and the tear of the macula appears secondarily.

Treatment. After some days rest, when maximal reapplication of the retina is attained, diathermy is applied as usual in the region of the superior tear. The external rectus muscle is then sectioned and one diathermic application, or two, is made near the macula and at its outer side for orientation, and light diathermy is applied in the macular zone. Puncture at the level of the superior tear. It is better in this case for the patient to lie in bed on the side of the operated eye, thus helping the passage of subretinal fluid from the macular region to the temporal region. Cure, 10-35 per cent.

FIG. 15—Total detachment with no visible tears. If the detachment is recent and the whole retina is visible, there are probably no tears, as, unless due to choroidal exudation, with so extensive detachment the tears would be very large. If the detachment is old, there might be small tears especially in the superior hemisphere. If part of the retina is not visible because of folds and prominences of the retina, it is possible that there are hidden tears.

Treatment. Rest and ophthalmoscopic examination to try and see the tears. If these are not visible, we are probably looking at a case of detachment of choroidal origin. In such case, focal sepsis should be excluded or treated with penicillin, pyretogenous cure, or cortisone. If the retinal prominence is not reduced, a puncture can be made to examine the fundus immediately or after a few hours. Cure, 10-30 per cent.

FIG. 16—Big tear near the periphery resembling a disinsertion. Severe case, owing to the size of the tear, which favors the rapid extension of the detachment and renders its reapplication difficult with rest.

Treatment. Barrier with flat diathermy and punctures between the tear and the posterior pole of the eye. Cure, 0-20 per cent.

FIG. 17—Detachment with tears spontaneously limited. Rarely, a detachment is limited by a line or barrier of chorioretinitis at the edge of the detached portion. The fundus appearance may remain unaltered for years.

Treatment. Hopeful with repeated examination or very light diathermy of the nondetached retina close to the watermark of chorioretinitis. Cure, 95 per cent.

FIG. 18—Very posterior tear with extensive retinal detachment. A rare type which though not associated with large tear is of very bad prognosis.

Treatment. After rest, diathermy and puncture at the zone of the tear, if possible; expulsion of the subretinal fluid and a small amount of vitreous. Cure, 10-30 per cent.

FIG. 19—Detachment with tear at the macula. Though the central vision is soon affected, the progress of the de-

tachment is slow owing to the small size of the tear.

Treatment. After rest, section of the external rectus; application of nonpenetrating diathermy, for orientation, near the macula at its external side. After localization of the macular region, further similar diathermy is applied, followed by puncture at the lower part. Cure sometimes requires some months, when subretinal fluid has become more dense. Cure, 50-80 per cent.

FIG. 20—Inferior detachment with tear in the medial temporal region. Owing to the tendency of the retinal fluid to descend, especially if the case is some weeks old and the tears are not large, one must look for these in the upper part of the detached zone.

Treatment. Rest, diathermy; if there is subretinal fluid, puncture with evacuation of the fluid by pressure or air injection. Cure, 40-90 per cent.

FIG. 21 — Inferior detachment with tear in the medial nasal region. Not so grave as the former because the papilla protects the macula.

Treatment. Same characteristics and therapy as for preceding case. Cure, 40-90 per cent.

FIG. 22—Large disinsertion of the ora serrata, of almost one third of the anterior insertion of the retina. The prognosis is poor as the communication between vitreous and retroretinal space impedes the application of the choroid to the retina by the reabsorption of the subretinal fluid.

Treatment. Rest for a few days; if there is no improvement, barrier with nonperforating diathermy and perforating diathermy from twelve to six o'clock at the site where the retina is closest to the choroid and where diathermic coagulations can be seen in the retina. Cure, 0-20 per cent.

FIG. 23 — Recent total detachment with disinsertion of four fifths of the ora serrata. Very rare case of sudden onset. Its rapidity was verified because the fellow eye was blind.

Treatment. Useless. Cure, 0.

FIG. 24 — Retinal detachment in the inferior hemisphere with several tears. This type, as all types of inferior detachment, may remain for a long time in this state, unrevealed to the patient.

Treatment. Rest. If improvement, diathermy in the region of the tears. If not, a diathermic barrier should be produced in the zone where the retina is near the choroid, recognized by seeing that the retina is coagulated by the diathermy. Puncture and evacuation of the fluid by pressure or air injection. Cure, 50-80 per cent.

FIG. 25 — Inferoexternal detachment with small tear. This may be unrecognized by the patient for a long time, as the detached retina corresponds to the amblyopic zone of the retina hidden by the eyebrow. The tears are very difficult to see if there are hemorrhagic opacities of the vitreous, which naturally tends to settle in the inferior pole of the globe.

Treatment. Same as for preceding case. Cure, 50-90 per cent.

FIG. 26—Extensive tear in the inferior equatorial region, resembling a disinsertion of the ora serrata. This type is generally the result of an advanced retinal degeneration or of cystic formations in the retina. It does not improve with rest owing to the large opening between the vitreous and the subretinal space.

Treatment. Diathermic barrier and punctures between the tear and the posterior pole in the zone which is closest to the retina. This is recognized by the coagulation observed when pressing

with the electrode when the current is on. Cure, 40-60 per cent.

FIG. 27—Disinsertion of the infero-external ora serrata. A type peculiar to the infantile and juvenile age periods and sometimes bilateral and symmetrical. It may also be caused by trauma and is of very slow evolution, taking sometimes many months to reach the macular region.

Treatment. Diathermic barrier as for the preceding case. Cures, 80-90 per cent.

FIG. 28—Old detachment of the inferior hemisphere with small tears or with no visible tears. In patients who are not good observers or in children, a detachment of this kind may exist for years without the patient consulting an ophthalmologist. Occasionally there are pigmented lines which prove that at previous periods the detachment had reached other limits.

Treatment. If no tears are seen, diathermic barrier, puncture and evacuation of the subretinal fluid by pressure or air injection. Cure, 30 per cent.

FIG. 29—Large detachment with inferior tear. Such a detachment arouses suspicion of a superior tear (in addition to the inferior one), which may be very difficult to see owing to its small size or because hidden by a fold. A history in which the patient observed a shadow by the side of the nose is confirmatory evidence of this. If the shadow was first observed above it is possible that there is an inferior tear only.

Treatment. Rest and diathermy of the tears with evacuation of the fluid after puncture at the inferior pole. Cures, 10-40 per cent.

FIG. 30—Retinal detachment due to albuminuric retinitis. This frequently occurs with the intense retinitis of pregnancy. Tears are usually absent.

Treatment. That of the general condition and termination of the pregnancy if necessary. Cures, 90 per cent.

FIG. 31—Detachment of the retina due to intense scleritis of the anterior pole. There were no tears. The detachment became extended greatly but resolved with subsidence of the scleritis.

Treatment. That of scleritis. Cures, 100 per cent.

FIG. 32—Retinal detachment accompanying detachment of the choroid. In certain cases of choroidal postoperative detachment (cataract, glaucoma) an associated and proximate retinal detachment may occur.

Treatment. Procure the healing of the corneal wound. Cures, 100 per cent.

FIG. 33—Retinal detachment accompanying a choroidal tumor. Transillumination and an exact measurement of the prominence of the retina covering the tumor is the key to the diagnosis, which may be difficult as sometimes the tumor is accompanied by a serous detachment.

Treatment. Coagulation if the tumor is small; if not, enucleation.

FIG. 34—Retinal detachment accompanying retinal angiomatosis (Von Hippel-Lagleyze). Almost all cases of this disease end with a detachment of the retina.

Treatment. In cases not very advanced, diathermy of the angiomas may influence favorably the disease, but once the detachment has appeared, the evolution is fatal.

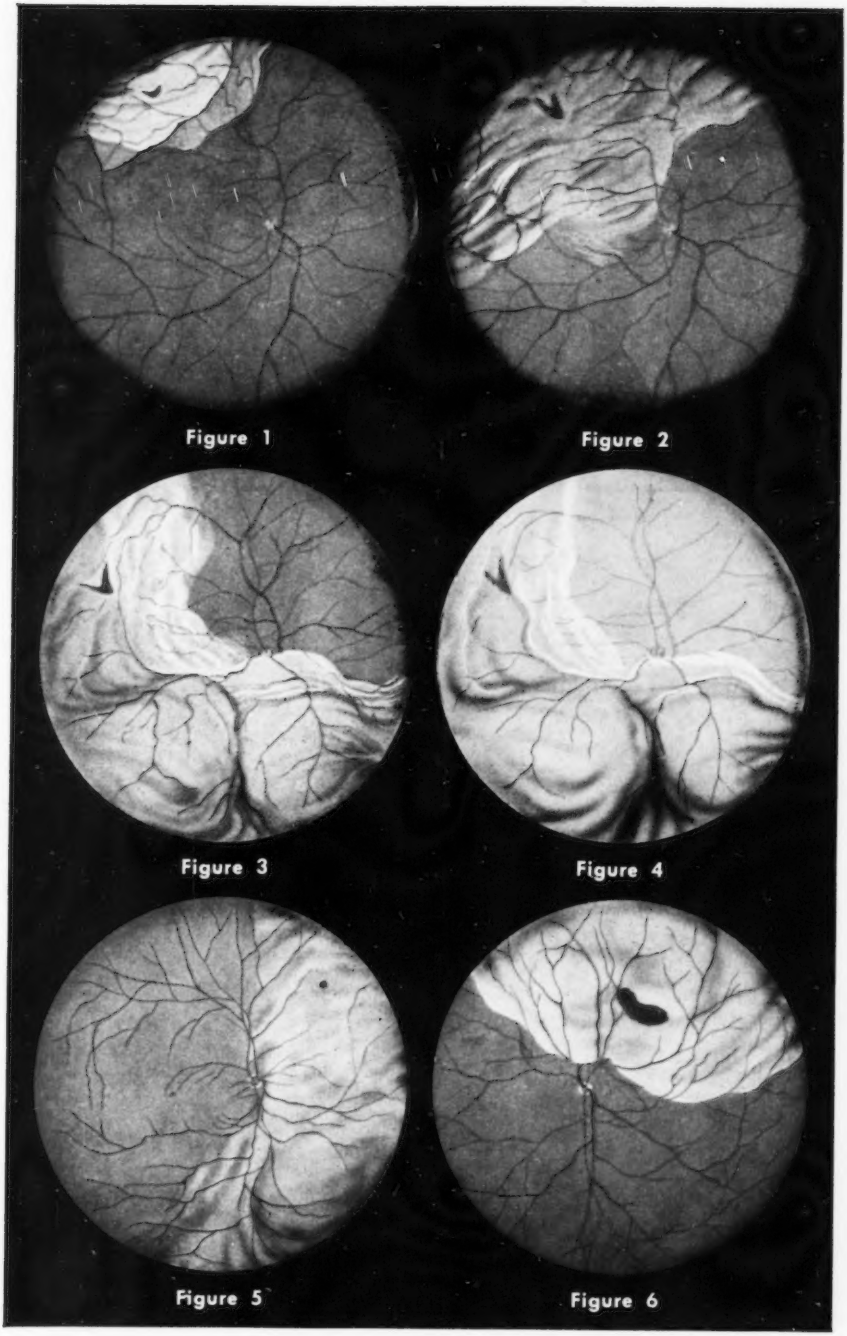

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Figure 3

Figure 4

Figure 5

Figure 6

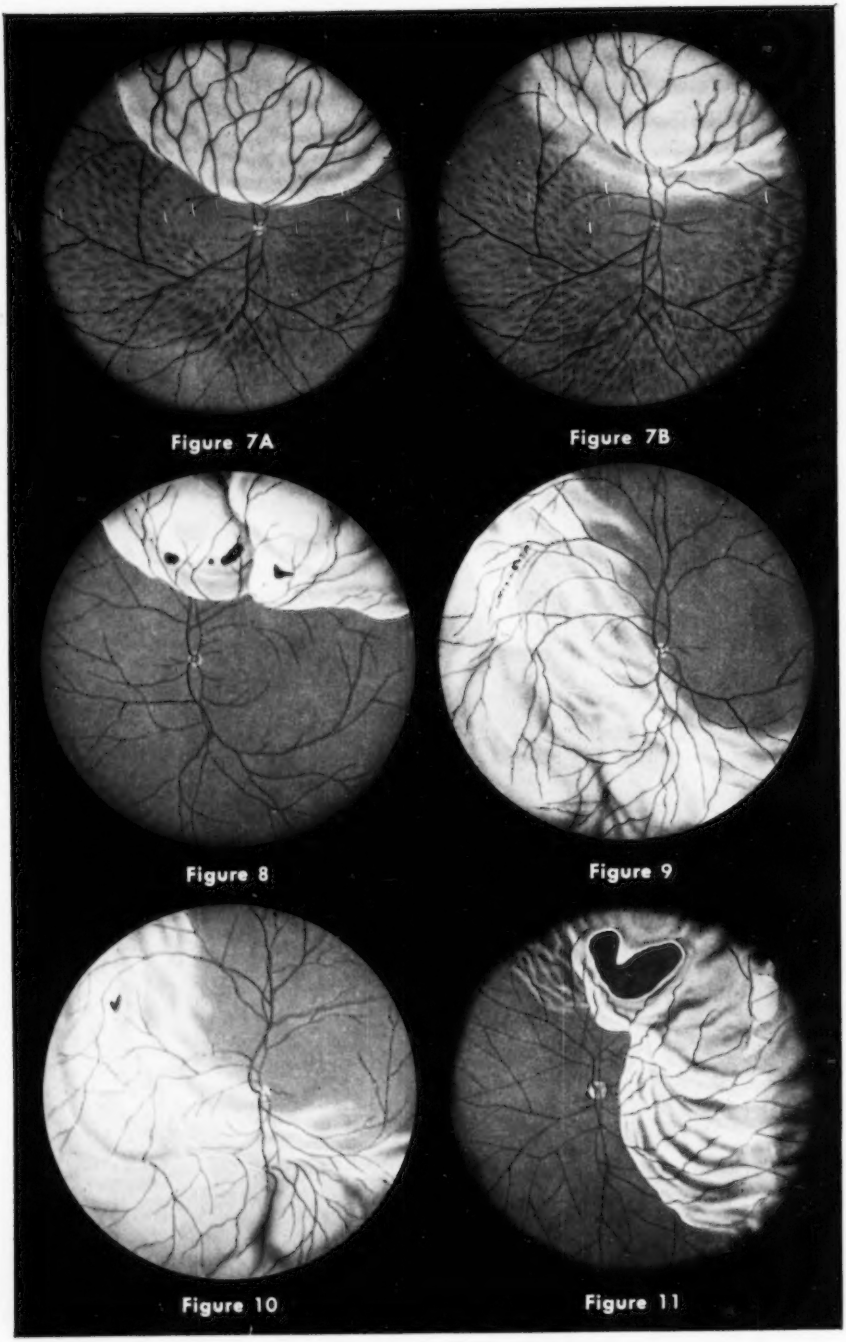

Figure 7A

Figure 7B

Figure 8

Figure 9

Figure 10

Figure 11

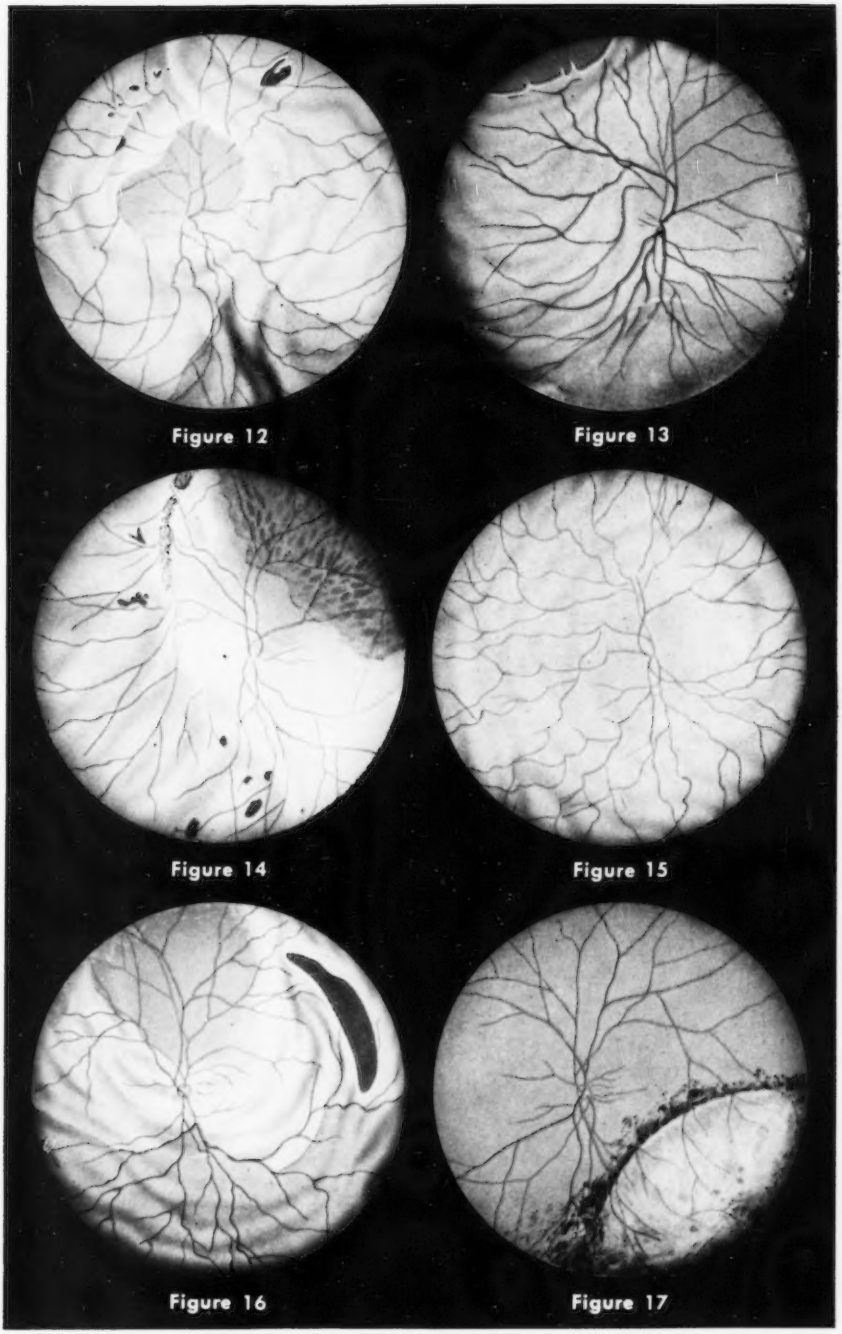

Figure 12

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Figure 16

Figure 17

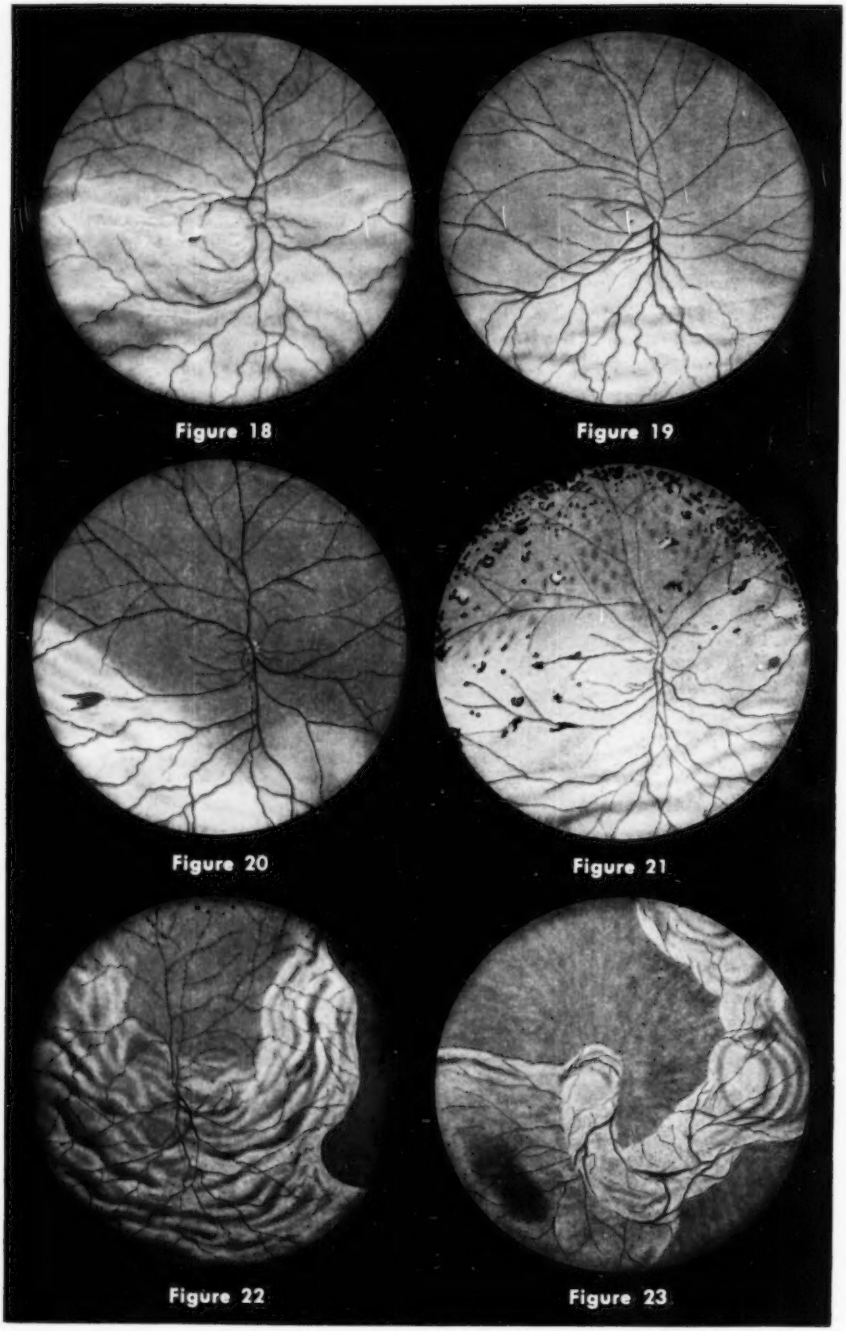

Figure 18

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Figure 20

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Figure 23

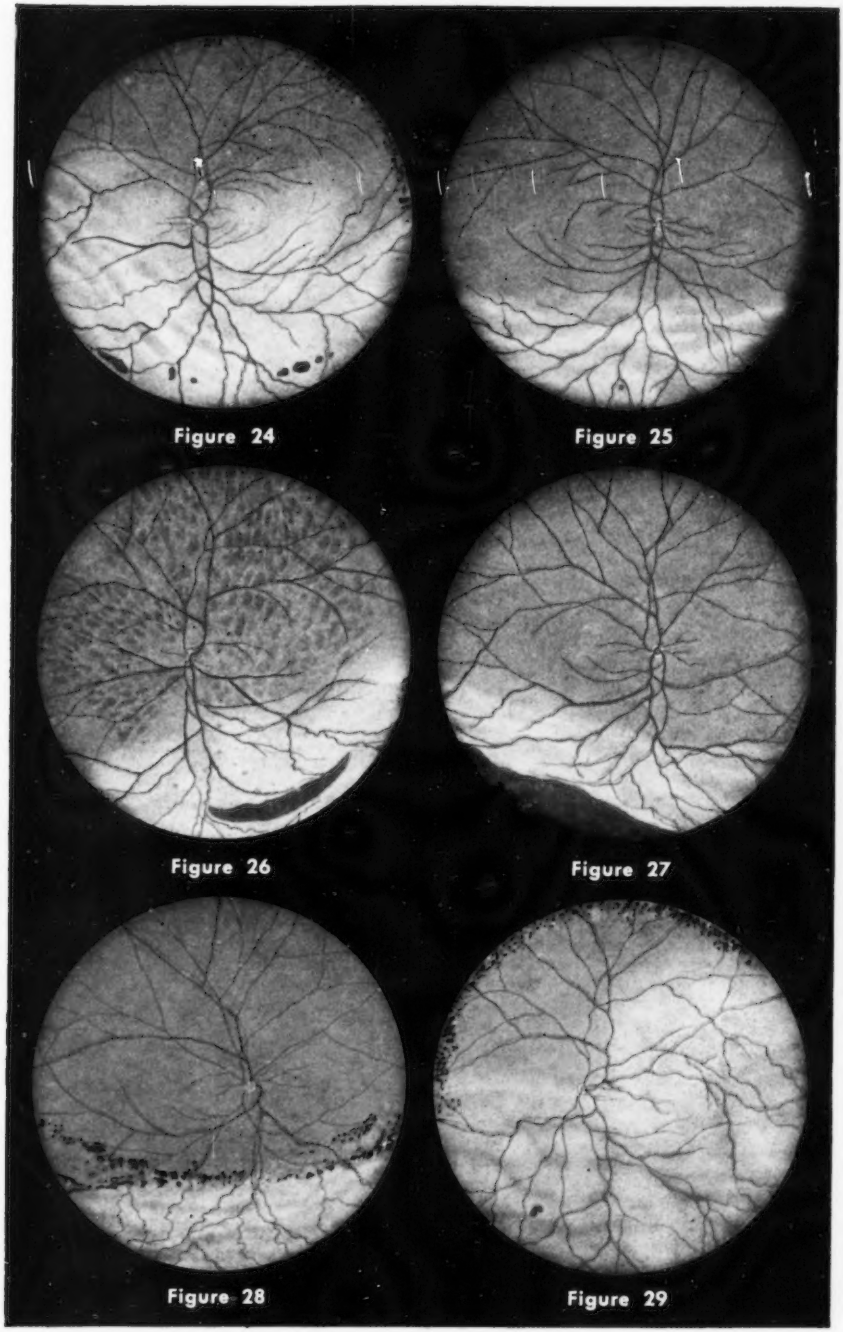

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Figure 28

Figure 29

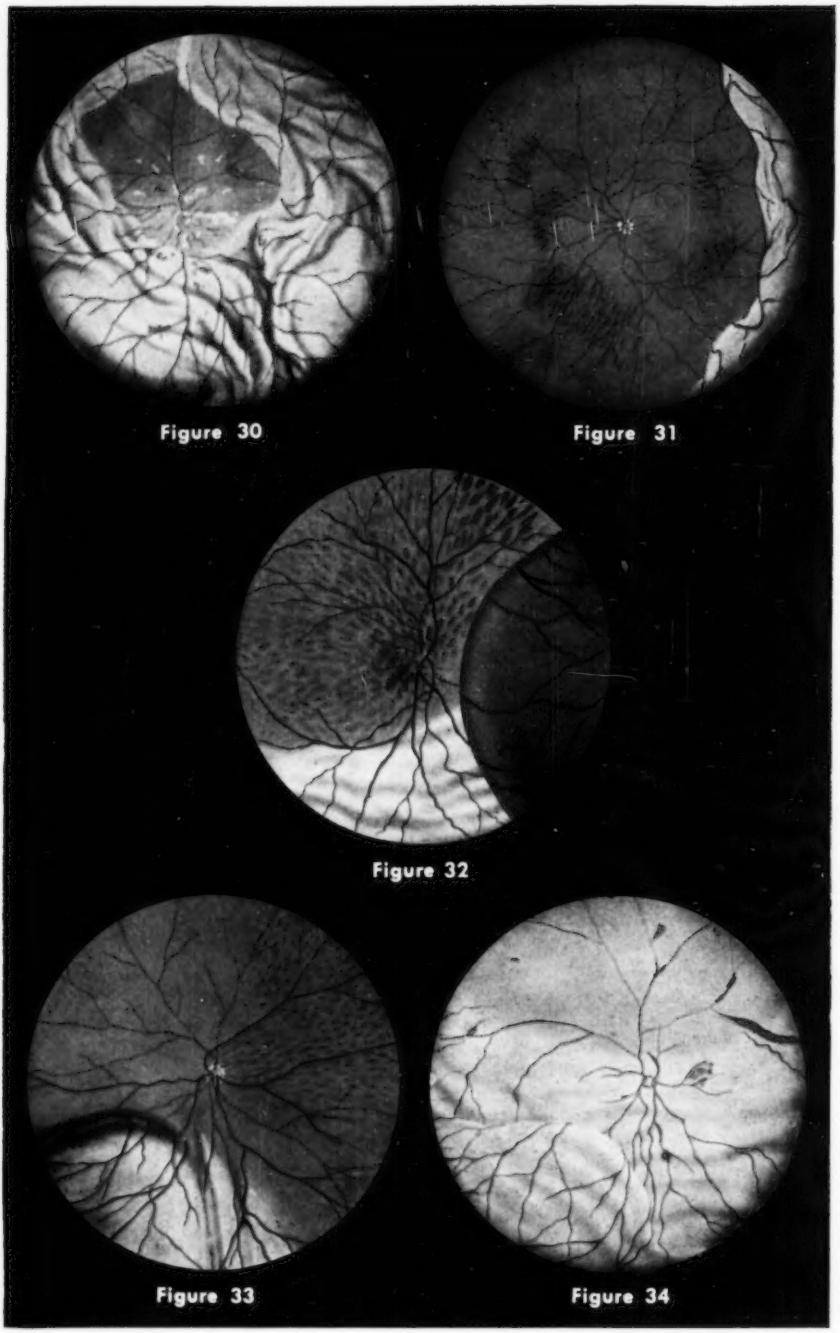

Figure 30

Figure 31

Figure 32

Figure 33

Figure 34

Symposium: Facial Injuries

ZYGOMATIC FRACTURE-DISLOCATIONS

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and
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EVEN to one who is not a semanticist the term "fracture of the zygomatic bone" is somewhat misleading and would seem more properly reserved for the less common incidents in which the continuity of the zygomatic bone itself is disrupted, usually in a comminuted manner (fig. 1, A). The malar bone is relatively massive and compact when compared to its attachments.[20] In the typical fracture-dislocation resulting from force directly applied to the prominence of the cheek, the zygomatic bone is detached and driven en masse downward, backward, and medially into the infratemporal fossa (fig. 1, B). The mere presence of this large depressed bone mass causes certain mechanical disturbance of jaw function. The fact that it forms appreciable parts of the lateral wall and floor of the orbit leads to ocular imbalance.

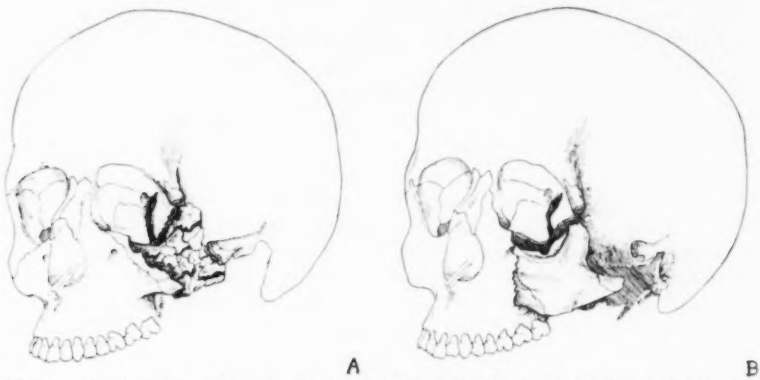

FIG. 1.—(A) Comminuted fracture of zygomatic bone; (B) Fracture-dislocation involving zygomatic compound with en masse displacement of large bone mass.

In the usual fracture-dislocation involving the zygomatic compound and resulting in separation of the zygomatic bone from its articulations, the superior line of separation is most often described as lying along the line of junction of the malar bone with the frontal bone and the great wing of the sphenoid (fig. 2). Such a situation could be expected to traumatize the structures that traverse the infraorbital fissure (the maxillary nerve and its zygomatic

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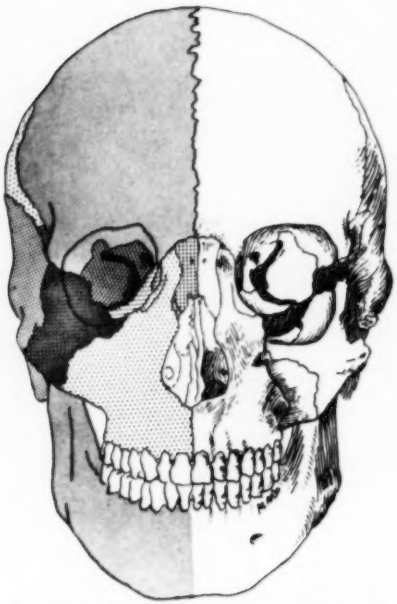


FIG. 2—Anatomic relationships in zygomatic fracture-dislocation as compared with the normal.

branch, the infraorbital vessels, and the vein connecting the ophthalmic vein with the pterygoid plexus) and produce maxillary nerve hypoesthesia or paresthesia and bleeding into the orbit. Although it is easy to conceive of the fracture extending into the great wing of the sphenoid, it is rather difficult to imagine it involving the roots of the lesser wing and the optic foramen. In fact, Pringle[17] claims that no one has ever been able to prove that an optic nerve lesion was caused by bony damage. On the other hand, Gordon and Macrae[6] reported a case of monocular blindness as a complication of malar fracture and theorized that such an occurrence could possibly be due to a spicule of bone impinging on the nerve.

An examination of the orbital floor and anterolateral wall of the maxilla, particularly when a difference in bony densities is demonstrated by a light

FIG. 3—Demonstration of thin bone in the region of the infraorbital foramen.

within the antrum (fig. 3), leaves one dubious as to the probability of the medial line of separation being along the zygomaticomaxillary suture. The thinnest bone in this region is the line of the infraorbital foramen of the maxilla. Crushing of this area accounts for the antral opacity that is nearly always present, since the lacerated mucosa causes the sinus to fill with blood (fig. 4); the hemorrhage may be large enough to fill the antrum and spill over into the nose via the maxillary ostium with resultant unilateral epistaxis. No further reasons for orbital and periorbital emphysema or for sensory disturbances of

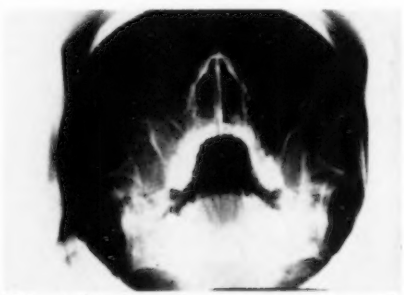


FIG. 4—Antral opacity accompanying zygomatic fracture-dislocation.

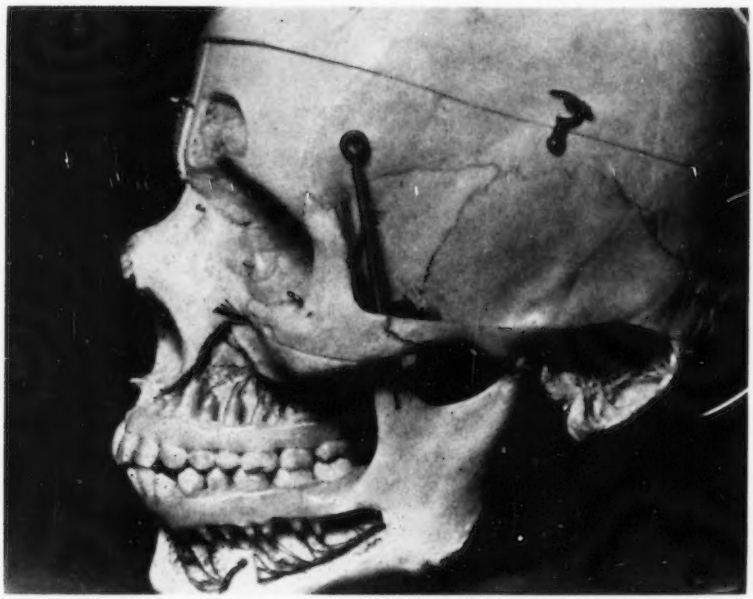


FIG. 5—Apparent strength of zygomaticotemporal suture line as compared with the adjacent zygomatic process of the temporal bone.

the infraorbital nerve need be sought if one concedes that the fracture line is across the foramen.

Examination of the skull (fig. 5) gives the impression that the serrated zygomaticotemporal suture is more stable than the portion of the zygomatic process of the temporal bone that lies immediately behind it. Whether there is a separation at the suture line or a fracture through the anterior part of the zygomatic process seems to be of little practical importance.

Pain accompanying movements of the lower jaw and mechanical blockage to its excursion are frequent findings[8] but are often of such small magnitude as to be overshadowed by other signs and symptoms and missed by the casual examiner. When there is almost complete locking of the jaw the attention of the inexperienced observer is usually direct-

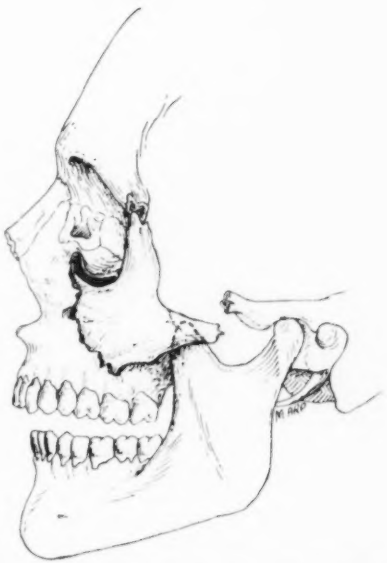


FIG. 6—Illustration of the zygomatic compound and subjacent coronoid process of the mandible.

FIG. 7.—(A) Extreme edema (24 hours after injury) preventing satisfactory palpation of orbital margins. (B) Same patient 72 hours later; orbital margins easily palpable.

ed toward the temporomandibular joint itself. If one realizes how the depressed zygomatic bone impinges upon the underlying coronoid process (fig. 6) and prevents rotation of the condyle about its two traverse axes[11] (one through the condyle and one through the mandibular foramen), he need not overly concern himself about the integrity of the joint. The immediate return of lower jaw motion after reduction of the fracture is sufficient proof that trismus is not due to joint damage or muscle spasm.

Palpation is probably the most useful procedure in the diagnosis of fracture of the malar compound; but when one is confronted with a hyperesthetic patient whose face is extremely swollen (fig. 7, A and B), the characteristic "step off" in the lower orbital rim, the irregularity of the lateral orbital border (fig. 1, B), and flattening of the malar eminence (fig. 8) may be impossible to demonstrate without heavy

FIG. 8.—Loss of malar prominence as a result of zygomatic fracture-dislocation.

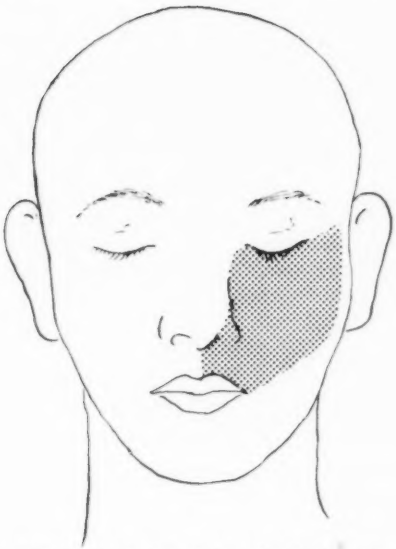


FIG. 9—Distribution of the infraorbital nerve.

sedation or general anesthesia. Except in unusual instances, unless a fracture can be positively diagnosed by other means, it probably does no harm to wait a few days for the swelling to subside so that the palpation can be more informative.

Anesthesia over all or part of the field supplied by the infraorbital nerve (fig. 9) may be difficult to evaluate even when present, as soft tissue trauma may produce sensory disturbances without underlying fracture.

The much-discussed diplopia accompanying fracture-dislocations is found in only about one tenth of all the cases. Its presence is a helpful diagnostic aid and has been attributed to injury to extraocular muscles and other orbital soft tissues, changes in orbital tensions due to edema, and altered direction of Lockwood's suspensory ligament.[9] It seems reasonable to assume that the last mentioned factor is of importance in the production of diplopia as the ligament's lateral attachment to the zygoma is carried downward, backward, and medially by the displaced bone. At the same time

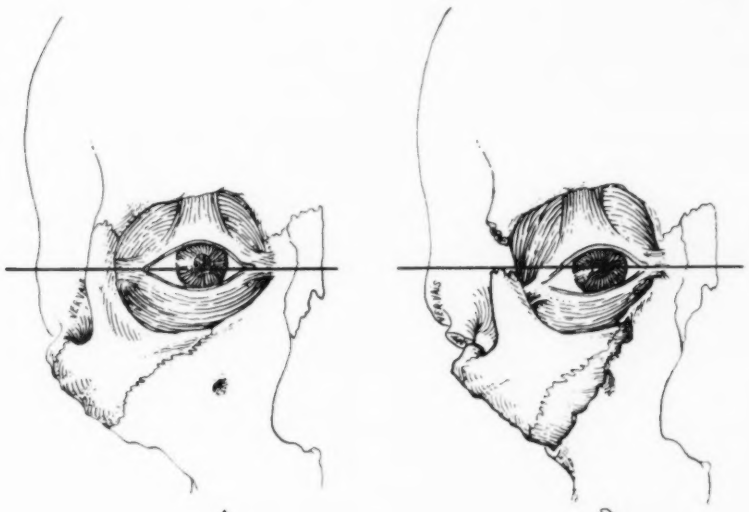


FIG. 10—(A) Normal plane of palpebral commissure. (B) Mechanism producing slanting commissure after displacement of the bony attachment of the lateral palpebral ligament.

the lateral palpebral ligament is carried away from its normal position so that the commissure assumes a slanting direction (fig. 10, A and B). When not obscured by edema of the lids, a slanting and narrowed commissure is more commonly present than is diplopia.

The more one deals with injuries to the zygomatic compound the more he becomes inclined to regard roentgenologic examination as only corroborative evidence and the less willing he becomes to lean heavily on it for absolute diagnosis or indication for treatment. Roentgenograms of all suspected fractures (fig. 11) are indicated not only for

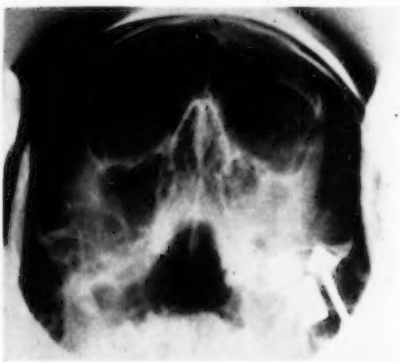


FIG. 11.—Roentgen demonstration of zygomatic fracture-dislocation. Note the clouding of the antrum and the lines of fracture.

what they can add to the impression gained from physical examination but also for medicolegal protection; however, the old cliche, "You can't operate on an X-ray film," remains valid. Too often even stereoscopic examination by a competent roentgenologist falls far short of being satisfactory. Nearly everyone can recall extreme distortions that were not evident on films, and other films that indicated remarkable misalignment when other examination revealed the dislocation to be too slight to merit treatment. Although antral opacity is almost always present as the result of hemorrhage from the sinus mucosa, the mucosa may bleed profusely from slight rents when bony displacement has been negligible. It is difficult to overemphasize the misinformation that roentgen examination may give; it seems wise for one to be rather definitely decided as to diagnosis and course of treatment before he has his first glance at the films and then to allow them only to suggest rather than to dictate.

TREATMENT

The treatment of fractures of the zygomatic compound may be resolved under the following headings:

Group I. Recent fractures that are easily reduced and easily maintained in proper position.

Group II. Fractures that are difficult to maintain in proper position.

Group III. Old fractures that cannot be reduced.

Those fractures that fall into group I can be satisfactorily handled by nearly any approach (fig. 12). Direct approaches through the overlying soft tissues,[13,14,18] and indirect approaches via the temple,[5] gingivolabial sulcus,[10] antrum[12] or nose[19,21] are in common use. Many operators appear anxious to avoid the direct approach because of the resultant scar on the prominence of the cheek. Others object to an intra-oral approach on the tenable ground that it is improper to introduce oral contaminants into the fracture field. The method of Gillies, whereby the elevator is passed between the temporal fascia which attaches to the upper border of the zygomatic arch and the temporalis muscle which runs deep to it, is a beautiful example of the application of surgical anatomy and breaks no rules of aseptic surgery. From observation of residents in training it appears to be the most difficult

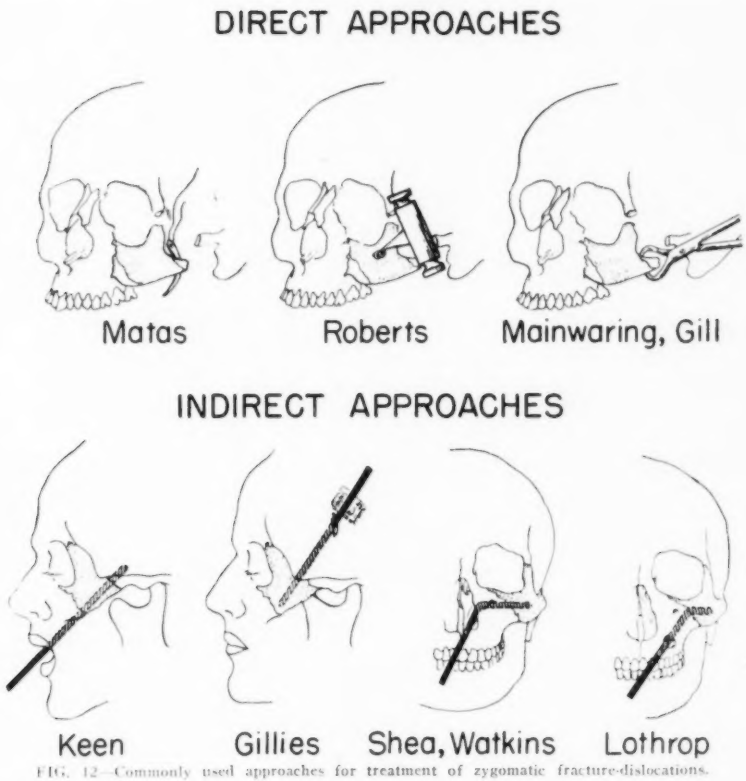


FIG. 12—Commonly used approaches for treatment of zygomatic fracture-dislocations.

approach to master, while the direct approach seems to be the least difficult. From the same source it has been learned that men who are making their first attempts to reduce fracture-dislocations are prone to commit common errors when using either the temporal or intrabuccal approach. In the first instance they are apt to miss the plane between the temporal fascia and muscles, either failing to pass the elevator when superficial to the fascia or passing it deep to the coronoid process or mandibular ramus when deep to the muscle (fig. 13). The first few approaches through the gingivolabial sulcus by the novice are almost invariably too far forward (fig. 14), the elevator being passed straight upward from the region of the premolar teeth or first molar tooth so that it passes anterior to the zygomaticomaxillary suture and lies in the soft tissues over the maxilla. Fortunately nearly all fractures will remain in proper position after reduction without resorting to any type of fixation.

The fractures of group II are often either more comminuted than usual or uncommonly impacted or bound down by fibrosis. The fracture may be easily reduced but impossible to maintain because normal supporting bone has been

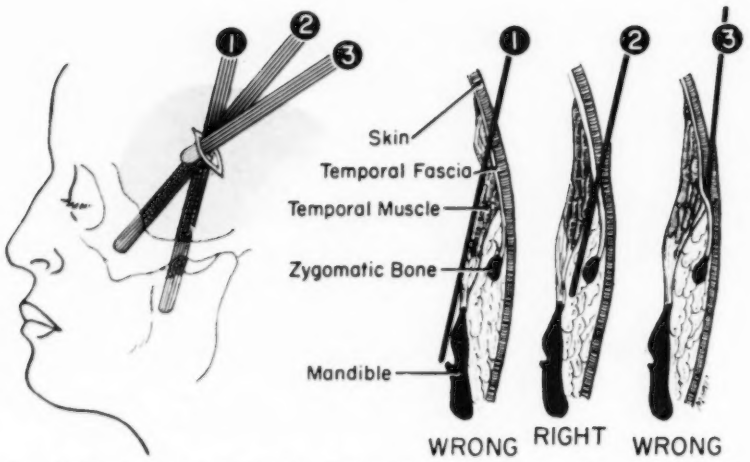
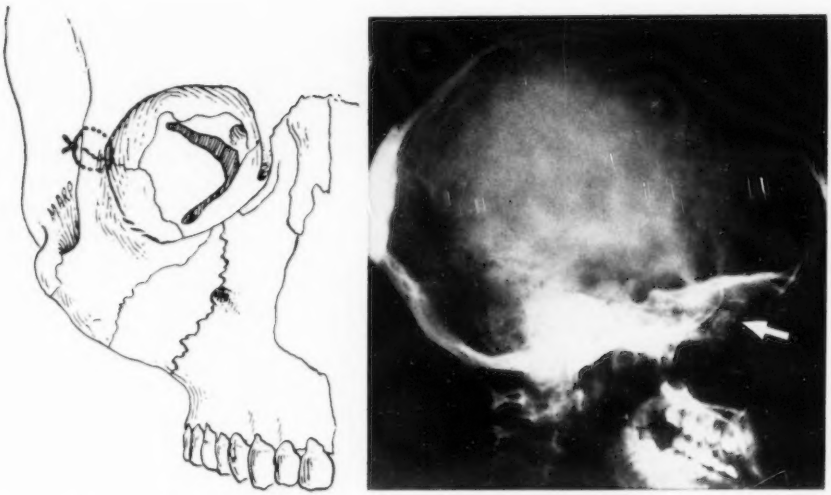


FIG. 13—Errors that are often made when using the temporal approach. Failure may be due to passing the elevator superficial to the temporal fascia (3) or passing it deep to the temporalis muscle (1).

crumbled. If the frontozygomatic junction has escaped extreme comminution, the dislocated bone can be kept in the optimum position by direct wiring at this point (fig. 15, A and B). Other methods of fixation include that of Ivy and Curtis[7] (fig. 16), in which a screw hook in the zygoma is attached to a metal arm extended from a plaster head cap or head band, and the procedure of Lothrop (fig. 17), in which fragments are held in position by firm packing of the maxillary sinus. Any of these procedures may be the preferable one under given conditions, but sinus packing left in place for several days does introduce the hazard of sinusitis, however slight the chance may be. It is almost surely less dangerous if a large nasoantral opening is provided at the time of packing.

FIG. 14—Common error made when using the intrabuccal approach. The elevator is passed so that it lies on top of the displaced bone.

Cases presenting undue impaction or fibrosis may allow some mobility of the depressed bone but not enough to permit adequate reduction. When impaction or fibrosis prevents immediate reduction, it may be obtained within hours or days by applying gradual elastic traction between a screw port in the bone and a



A B

FIG. 15—(A) Usual method of maintaining reduction by interosseus wiring. (B) Reduction maintained by interosseus wiring. The use of heavy wire is usually unnecessary; 32-gauge steel wire, which is barely visible on roentgen examination, was used in this case.

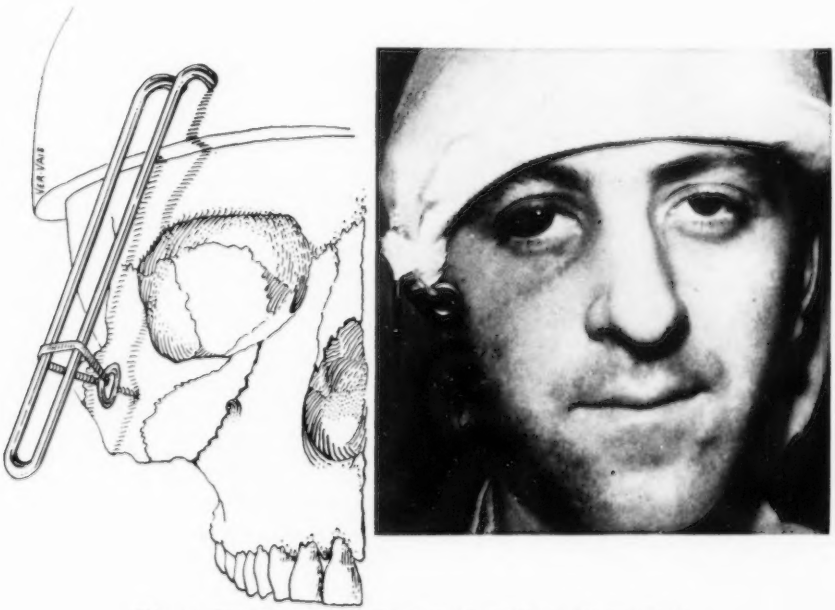


FIG. 16—Fixation of fracture-dislocation by method of Ivy and Cutts.

FIG. 17.—Intra-antral packing used to maintain position.

fixed arm. The antral approach of Lothrop may help to overcome a firm impaction by applying force from a different direction than could be brought to bear from a temporal, intrabuccal or direct approach, but usually it does not offer much additional advantage when the dislocated bone is involved in fibrosis.

A fracture that absolutely resists reduction is nearly always one that has been neglected long enough for firm bony union in false position to take place. These cases often present themselves on the ophthalmologic service with the complaint of double vision which may not have become troublesome until several weeks after the injury. As will be pointed out later, we are a little more stubborn than most others when it comes to admitting that a fracture cannot be reduced by some method. When that admission is finally made, two courses for correction are open: osteotomy with repositioning or reconstruction of the orbital floor and malar eminence with modeled or diced bone or cartilage (fig. 18).[1-4,15,16] Visits to various clinics leave the impression that the consensus is that the latter course is the simpler and safer.

We hope to avoid leaving the impression that our routine for the diagnosis and treatment of zygomatic fractures is the only one or by any means the best one. It has evolved hit-or-miss over the years and may seem efficient to us only because we are accustomed to it.

The patient with a suspected fracture usually stops for roentgenograms on his way from the admitting office. When first seen by the physician who is to treat him, an attempt is made to put him at his ease and assure him that he will not be hurt. A few minutes conversation will usually elicit important information and cause even a child or extremely nervous adult to permit later examination that he would otherwise resist. The time of the injury, the point of the blow, a description of a "funny feeling" in the lip, a complaint of double vision, or information concerning unilateral epistaxis will often be volunteered while the patient is overcoming his excitement and learning that he is not faced by a pain-dealing monster. During this "cooling-off" period ample time is allowed for observation of swelling, ecchymosis, loss of malar prominence, and slanting commissure.

Examination for diplopia in all directions of gaze followed by gentle stroking

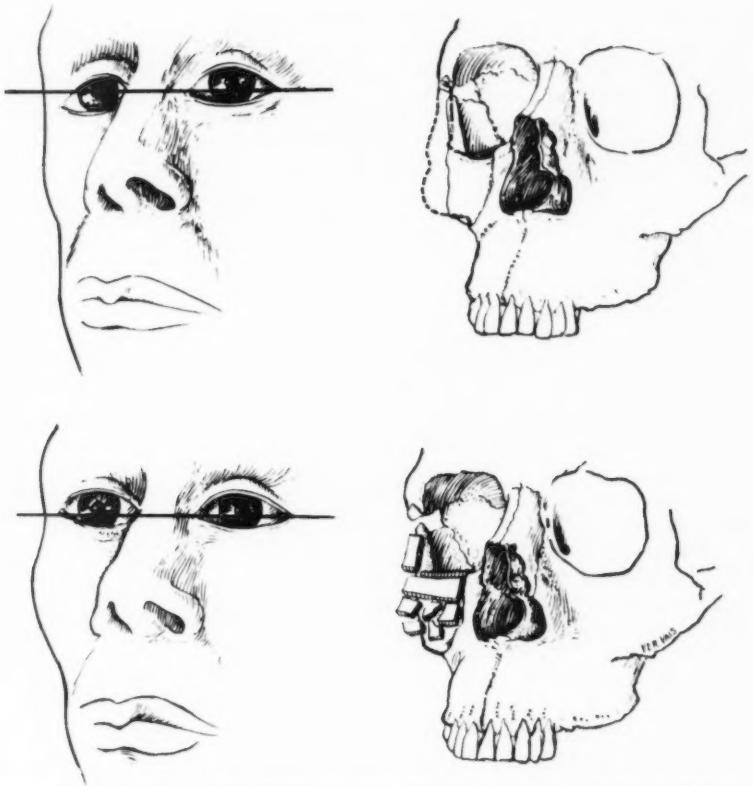


FIG. 18.—Method of restoring contour of cheek and orbital floor with grafts of bone or cartilage.

of the upper lip and possibly antral transillumination lets the patient begin to believe that he can be examined without having to suffer.

We often allow the patient to palpate his own lower orbital rim before attempting to do so ourselves. On numerous occasions patients, including several children, have discovered and proudly pointed out the characteristic "step off." Very rarely, edema is so severe that adequate palpation of the lower and lateral orbital rims and the malar prominence is unsatisfactory. At such times the patient may be examined under sedation or general anesthesia or allowed to wait one or more days until edema subsides. We prefer the latter course unless contraindicated. Sometimes intraoral palpation will reveal the dislocated bone when it cannot be felt through the skin. By this time the examiner is, as a rule, fairly well decided as to diagnosis of fracture and intended reduction and is ready to consider the roentgenographic findings.

If a fracture demanding treatment is present, reduction by the temporal approach is attempted first and is nearly always successful; if this fails, an intra-

buccal attack is made. The occasional fracture resisting these procedures is subjected to a direct approach. If the fracture is a fresh one and is still in unsatisfactory position, a Lothrop approach to break up the impaction may be made or elastic traction may be applied.

In the injury where fibrosis is expected because of the time elapsed since it was inflicted, immediate reduction is always tried before the elastic traction is applied. This does little harm and at times produces unexpected but welcome results. On one occasion a member of our staff amazed himself by easily reducing a fracture that was nearly two months old.

When the time elapsed between trauma and treatment is sufficient to make reduction obviously of no avail and reconstruction is indicated, we prefer to use either carved or chipped cancellous bone, or both, to restore the height of the orbital floor and rebuild the malar eminence.

SUMMARY

Zygomatic fracture-dislocations are among the most common facial bone injuries and usually result from a direct blow. Because of early intensive swelling of the adjacent soft tissues, recent depressed fractures may go unrecognized unless they are especially considered. They are characterized by depression of bone into the maxillary sinus and infratemporal fossa; the displaced bone mass results in disruption of the bony orbital wall, mechanical disturbances to jaw function, trauma to the infraorbital nerve, tearing of the sinus mucosa, and loss of normal cheek contour.

The objectives of treatment are the replacement of the zygomatic bone in its proper position, the relief of neurologic and temporomandibular symptoms, and the restoration of normal facial proportions. There are numerous methods of attaining these objectives. The procedures used in any given case will depend upon the type of fracture, the time elapsed since the injury, and the personal experience of the surgeon. Early reduction is desirable since rapid fixation of the displaced bone complicates an otherwise relatively simple operation.

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Symposium: Facial Injuries

* * *

FRACTURES OF THE MAXILLA

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FRACTURES of the maxilla are divisible into four groups: (1) fractures which cause complete separation of the upper jaw from the rest of the skull; (2) horizontal fractures of the maxilla associated with one or more fractures of the palate and alveolar process; (3) unilateral fractures of the upper jaw; (4) simple fractures of the upper alveolar process.

This systematic arrangement of fractures involving the upper jaw is a convenient classification because every fracture of the maxilla will fall into one or another of these four groups. Moreover, each group of fractures requires a specific form of treatment.

Before discussing each of these four groups of fractures which involve the upper jaw, I would like to emphasize the fact that nearly every fracture of the jaw in which the fragments are displaced causes some disturbance of occlusion, and unless the teeth can be brought into normal relationship, the fragments will not be restored to their original position. Nothing serves as a better guide to the proper position of the fragments than does occlusion of the teeth.

Prior to actual reduction of the fracture, debris and any loose pieces of bone which unquestionably would form sequestra should be removed. Furthermore, stress should be placed on the importance of extracting every tooth, the root of which is exposed in the line of fracture, unless the tooth is absolutely necessary for fixation of a bony fragment. In my experience, removal of such teeth is an extremely important step in prevention of abscess and other complications attributable to infection.

The hurried reduction and immobilization of fractures of the upper jaw, with little thought being given to the selection of methods of treatment most suitable in each case, often produces poor functional and cosmetic results. I believe that even for simple fractures, there is no harm in waiting a few days before undertaking treatment. As a matter of fact, treatment should be delayed until satisfactory roentgenograms of the bones of the face can be obtained and until the most desirable method of treatment can be determined. Complicated and serious fractures of the upper jaw often require a great deal of preoperative planning and sometimes preparation of special appliances for reduction and fixation. Under such circumstances, a few days may be necessary to complete the plan of treatment.

As will be discussed later, some fractures of the maxilla are associated with fractures of the cribriform plate of the ethmoid bone. If such a fracture of the cribriform plate is associated with a tear in the dura, cerebrospinal fluid usually drains from the nose for several days following the injury, and the dan-

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ger in these cases is the possible development of meningitis. Every patient with a serious maxillary injury should be examined for possible cerebrospinal rhinorrhea because the diagnosis of an associated ethmoid fracture is of the utmost importance in determining the subsequent treatment. While many surgeons believe that there is no harm in going ahead with immediate treatment for fractured facial bones associated with a fractured cribriform plate, it is my opinion that patients with such injuries should be left alone for ten to fourteen days following the injury. Just as good therapeutic results can be obtained in cases of fracture of the maxilla if treatment is delayed a week or two as can be obtained by immediate reduction and immobilization following the injury. Moreover, attempts at reduction of fractures of the maxilla associated with a fractured cribriform plate during the first week or two following the injury may disturb the fracture of the ethmoid bone and invite infection of the adjacent meninges from the nasal cavity. When the diagnosis of ethmoid fracture with cerebrospinal rhinorrhea has been established, a neurosurgeon should be consulted. In some cases, spicules of bone may protrude into the overlying brain tissue, and under these circumstances an open operation to remove such spicules is occasionally indicated. It has been my experience that by ten to fourteen days following such an injury, the fracture of the cribriform plate becomes sealed off by fibrous tissue and the cerebrospinal rhinorrhea stops. Thereafter, one may proceed with the necessary treatment of the fractured maxilla. The use of antibiotics is of the utmost importance during this ten-day to fourteen-day interval of waiting. Moreover, while meningitis is the most important complication to be avoided following a fracture of the cribriform plate, one must not overlook the possibility of a brain abscess developing or, subsequently, of pneumocele developing.

The reduction of fractures of the upper jaw in general is based on mechanical appliances which restore the teeth to normal and their original occlusion. Even though the fragments of the upper jaw may be freely movable, it usually is not possible to manipulate them into correct position. On the contrary, one must depend on traction with rubber bands to secure satisfactory reduction of the fracture and normal occlusion of the teeth. Gradual, continuous pull of rubber bands, when applied in the right direction, almost invariably will restore a displaced upper jaw to its proper position, whereas the ultimate result would be more or less a failure if one attempted manually to replace the fragments of a fractured maxilla in accurate alignment.

FRACTURES WHICH CAUSE COMPLETE SEPARATION OF THE UPPER JAW FROM THE REST OF THE SKULL

It is advisable to consider three forms of fractures which cause complete separation of the maxilla from the rest of the skull (fig. 1). As illustrated in figure 1*a*, the first and most common type is the horizontal or transverse fracture of the upper jaw, in which the greater portion of the maxilla is detached as a free segment. The second type (fig. 1*b*), not infrequently encountered, is the pyramidal facial fracture (so-called because it assumes the form of a pyramid) which extends upward through each maxillary sinus to the ethmoid region and base of the nose. In this form, the loose fragment is composed of the entire maxilla and the nasal bones. Pyramidal facial fractures often are associated with a depressed or comminuted fracture of one malar bone. Of rare oc-

currence is the third type (fig. 1*c*), which may be described as a transverse facial fracture passing through the base of the nasal and ethmoid regions and across the orbits to the zygomatic arches. This fracture is of such character that the upper jaw, malar and nasal bones constitute one complete displaced structure. As would be expected, the last type of fracture is extremely uncommon because it almost invariably is present in conjunction with depressed fractures of both malar bones. One should not overlook the fact that two, or all three, of these fractures may exist together in the same case. Moreover, fractures of the malar, nasal and ethmoid bones are frequently encountered in connection with severe fracture of the upper jaw.

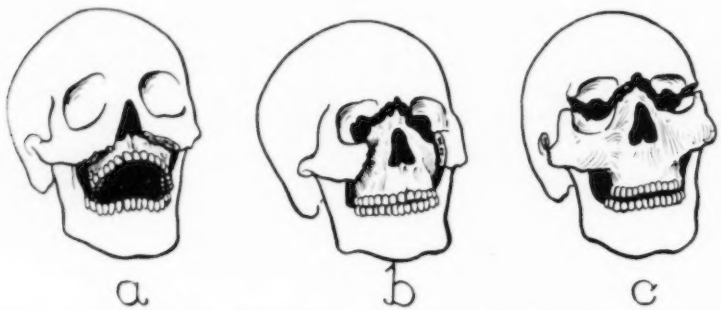

FIG. 1.—(*a*) Horizontal maxillary fracture; (*b*) pyramidal facial fracture; (*c*) transverse facial fracture.

Although the three fractures which have been described possess definite anatomic differences, the objective intraoral findings are often indistinguishable. Clinically, the upper jaw is loose and movable and more or less displaced, so that the teeth do not occlude properly. However, in a horizontal maxillary fracture, there is a tendency for the upper jaw to be forced upward and backward, with a resultant open-bite type of deformity. Pyramidal facial and transverse facial fractures, on the other hand, are likely to cause downward displacement of the upper jaw, with varying degrees of malposition between the upper dental arch and the lower teeth. In such instances, roentgenograms frequently reveal a space several millimeters wide between the upper border of the nasal bones and the glabellar portion of the frontal bone, and there is frequently an associated fracture of the cribriform plate of the ethmoid bone, with cerebrospinal rhinorrhea.

In spite of the fact that the therapeutic management of the three types of fractures under consideration is essentially the same, their early clinical recognition through roentgenographic studies is distinctly advantageous. The preoperative diagnosis of the type of fracture present is of value in ascertaining the gravity of the injury, since fractures of the second and third type are frequently associated with fracture of the cribriform plate of the ethmoid bone, a complication that could lead to fatal meningitis.

In treating fractures of the upper jaw associated with fracture of the cribriform plate of the ethmoid bone, any injury of the overlying soft tissues of the face should, I believe, be taken care of as soon after the injury as is feasible.

However, as previously discussed, I believe it is advisable to defer treatment of the fractured maxilla for from ten to fourteen days after the injury, because any manipulation of this bone may disturb the fracture of the ethmoid bone and induce meningitis.

As applies to all fractures, treatment of injuries of the facial bones which are associated with complete separation of the upper jaw from the rest of the skull consists first in reduction and later in immobilization of the parts involved.

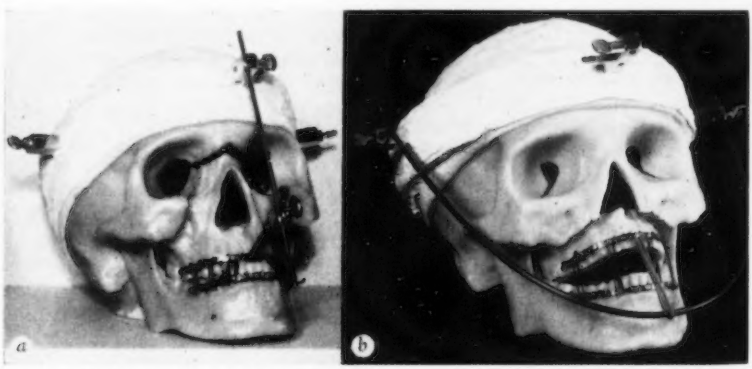

FIG. 2—Method of reducing horizontal maxillary, pyramidal facial and transverse facial fractures by intermaxillary elastic traction. (*a*) Method of pulling jaw forward by elastic traction; (*b*) method of pulling jaw downward by elastic traction.

The reduction by elastic traction of any of the three types of maxillary fracture under consideration requires, first, application to the upper dental arch of a hooked arch bar, which is wired securely to each upper tooth (fig. 2). Next, a similar arch bar is wired to the teeth of the lower jaw. Then rubber bands stretched between the hooks of the upper and lower arch bars gradually pull the teeth into occlusion and, in turn, the upper jaw into proper alignment. If the maxilla is displaced backward, direct anterior traction becomes necessary in order to effect complete reduction of the fracture. This can be accomplished by stretching a rubber band from the upper arch bar to a rod attached to a plaster head cast (fig. 2*a*). Such traction with rubber bands will give sufficient forward pull to bring the jaw into the desired position. In many cases of horizontal maxillary fracture, the upper jaw is displaced upward and backward to a marked degree, and very often the loose fragment is impacted in this position. Under such circumstances, strong downward traction is required to bring the teeth into occlusion. Here, a strong rubber band stretched from the upper arch bar to a curved rod, which passes downward anteriorly to the chin from the head cast, will give the desired downward pull on the upper jaw (fig. 2*b*).

After reduction of the fractured upper jaw and restoration of normal dental occlusion, some form of immobilization becomes necessary to maintain the upper jaw in proper position until healing has occurred. For fixation, I have tried many methods which have been advocated in the literature, and I believe that I can obtain the most satisfactory results by the use of traction wires inserted through the cheeks, as was first described by Federspiel (figs.

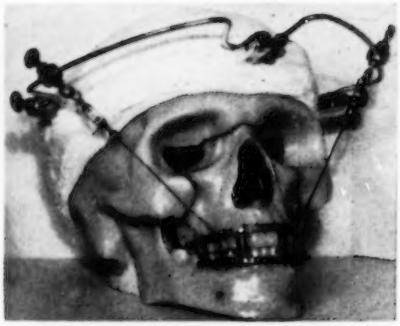

FIG. 3—Method of fixation of horizontal maxillary, pyramidal facial and transverse facial fractures by traction wires inserted through the cheeks and attached to a plaster head cast.

3 and 4). In the application of such traction wires, I employ two double strands of 26-gauge stainless steel wire. Each strand is attached to the upper arch bar in the bicuspid region, is passed directly through the cheek and is fixed to a plaster head cast above. These traction wires permit a direct upward pull on the upper jaw. They offer a form of immobilization which can be maintained for an indefinite period and which leads to strong, bony union of the maxillary fracture. In most instances, I maintain fixation with traction wires for three to four weeks.

FIG. 4—Patient with horizontal fracture of the maxilla immobilized with traction wires passed through the cheeks. Adjustable hooks on the head cast permit changing the position of the wires so that there is no lateral pressure of this wire on the soft tissues of the cheek, thereby preventing scarring and dimpling of the cheeks.

When one employs traction wires which are attached to a fixed post on the head cast, there is a strong probability of producing in each cheek a linear scar, which may become retracted to form a dimple. The explanation of such scarring is based on the fact that a straight line between the upper arch bar and a fixed post on the head cast passes through the skin of the cheek at a very definite point. Since it is impossible for one to determine the exact position of this point in advance, when the traction wire is inserted through the cheek, it usually emerges several millimeters away from the desired point. Consequently, when the wire is made tense, it pushes the soft tissues aside and the subsequent inflammatory reaction cuts through the skin to produce a linear scar. However, by the use of adjustable hooks (figs. 3 and 4) attached to the head cast, I have been able to overcome this undesirable scarring of the cheeks. I employ two hooks, which slide from side to side on a curved rod fixed to three metal posts incorporated in the plaster head cast. These hooks also can be made to swing anteriorly or posteriorly as desired. Through this range of movement, the hooks can be adjusted so as to engage the traction wires in any plane in which they emerge from the cheeks. This prevents any lateral pressure of the traction wires on the soft tissues and, in turn, any tendency of these wires to cut through the cheeks.

It is not uncommon for the upper jaw, when completely detached from the rest of the skull, to be displaced downward, and one finds that mere manipulation does not elevate the jaw into proper

position. This is because of the impingement of spicules of bone along the line of fracture. However, the upper jaw can be brought upward by elastic traction, rubber bands being inserted between the hooks on the plaster head cast and the traction wires passing through the cheeks. It should be stressed, however, that if such elastic traction is employed, the rubber bands should be replaced with wires as soon as the maxilla has been elevated sufficiently.

I would like to consider briefly the use of plaster head casts because many surgeons have tried and discarded them for some form of adjustable headgear. It has been my experience that plaster head casts give the most satisfactory results. However, if not properly constructed, they will move and will fail to hold the traction wires securely in place. The proper construction of a plaster head cast cannot be discussed here, but the fact can be mentioned that, in stabilizing a plaster head cast, I have found it advisable to shave the back of the patient's head up to and over the occipital prominence. A strip of sponge rubber about ¼ inch (about 0.6 cm.) thick and measuring in width and length about 1 by 4 inches (about 2.5 by 10.2 cm.) can then be applied to the shaved skin on the back of the head and held in place with a narrow strip of adhesive tape. The head cast then is built over this piece of sponge rubber. The friction provided by the rubber that lies between the plaster cast and the head is extremely helpful in preventing displacement of the head cast.

It is desirable to relieve pressure of the head cast anteriorly when traction wires inserted through the cheeks are employed, for these wires tend to tip the front of the plaster cast downward, with the result that an undue amount of pressure is applied to the forehead. In order to obviate this form of pressure, a strip of felt or sponge rubber ¼ inch (about 0.6 cm.) thick is applied to the forehead with adhesive tape before the head cast is constructed. After the plaster cast has been completed, this piece of felt or rubber is removed, a space remaining between the cast and the skin of the forehead. By this procedure, pressure sores attributable to tissue necrosis in the region of the forehead are prevented.

A metal band equipped with three posts is incorporated in the cast. This band does away with the necessity of installing on the plaster cast makeshift appliances to which traction wires for immobilization of the upper denture are attached; the posts permit the use of adjustable hooks for holding the traction wires.

Fractures of the facial bones, with complete separation of an edentulous upper jaw, at times, offer considerable difficulty in reduction. It is not always possible to reduce such fractures completely, but this is usually of little consequence provided the upper jaw can be thoroughly stabilized to bring about good bony union of the fracture line. It has been my experience in such cases that traction wires which are attached to the upper denture or to a vulcanite splint give very satisfactory immobilization for the maxilla and aid in securing the desired bony union.

HORIZONTAL FRACTURES OF THE MAXILLA ASSOCIATED WITH ONE OR MORE FRACTURES OF THE PALATE AND ALVEOLAR PROCESS

When a transverse fracture of the maxilla is associated with one or more fractures of the palate, it is necessary that the fragments of the palate and alveolar process be brought into correct apposition before the upper teeth are returned to normal occlusion with the lower dental arch by elastic traction.

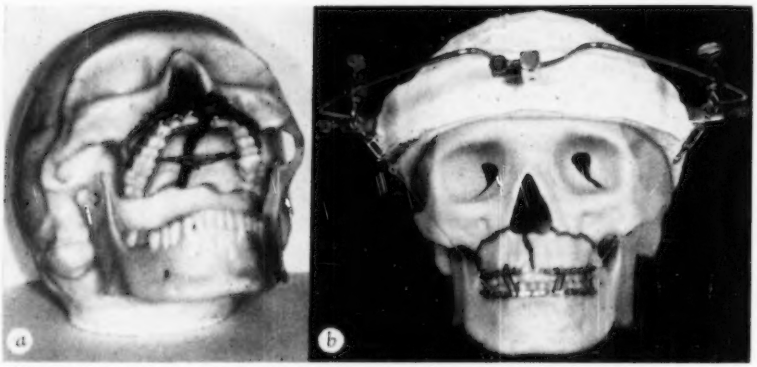

FIG. 5—Horizontal fracture of the maxilla with a fracture through the median line of the palate. (*a*) Fragments approximated by rubber band traction attached to hooks, as illustrated; (*b*) immobilization of the fragments of the upper jaw by traction wires attached to the full lower arch bar.

The most common type of palatal fracture is the one which occurs in the median line. In cases of this type (fig. 5), I apply a hooked arch bar to the upper teeth and, subsequently, divide the arch bar at the site of the median line fracture. On each side, a hook is attached to each segment of the arch bar so as to emerge lingually in the interproximal space between the second bicuspid and first molar tooth. A rubber band then stretched between these two hooks will gradually bring the two halves of the palate and alveolar process into proper approximation (fig. 5*a*). One can then proceed with reduction of the transverse maxillary fracture by the use of vertical traction with rubber bands, as has been described (fig. 5*b*).

In multiple fractures of the palate, associated with a transverse maxillary fracture (fig. 6), there is often but a single tooth present in a fragment. When such a tooth is to be used for stabilization of the fragment, I believe that it is preferable to adapt an orthodontia molar

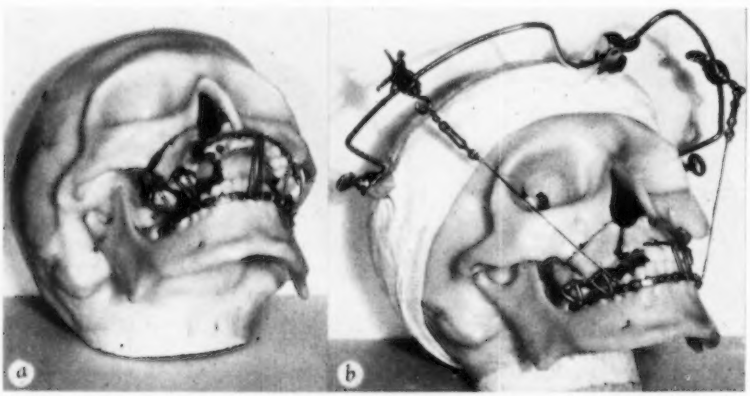

FIG. 6—Horizontal fracture of the maxilla with multiple fractures of the palate and alveolar process. (*a*) Restoration of dental occlusion by intermaxillary elastic traction; (*b*) immobilization of the upper maxillary fragments by traction wires attached to an arch bar wired to the lower teeth.

FIG. 7.—Unilateral fracture of the upper jaw. In this type of fracture, the loose fragment of bone is displaced downward and inward.

band to the tooth rather than to apply a wire around the neck of the tooth. Such a molar band, when securely attached to the tooth, cannot be displaced and its buccal sheath serves as an excellent attachment for intermaxillary rubber bands or wires. Where there are several teeth in a fragment, it is preferable to employ a portion of an arch bar, wiring it to each tooth in the fragment. Then vertical rubber bands, stretched from the upper molar bands or arch bar segments to a hooked arch bar below, will gradually bring the upper teeth down into normal occlusion with the lower dental arch (fig. 6*a*). Since there is no full upper arch bar to which traction wires can be attached, it is necessary that they be applied to the lower arch bar (figs. 5*b* and 6*b*). This procedure firmly fixes the mandible and, in turn, the fragments of the upper jaw are immobilized. Fixation is maintained for about four weeks.

UNILATERAL FRACTURES OF THE UPPER JAW

Usually, a unilateral fracture of the upper jaw is the result of a blow directed from the side and, consequently, the loose fragment of bone is displaced downward and inward (fig. 7). Moreover, such a fracture is commonly associated with a depressed fracture of the malar bone on the same side, and not infrequently with a fracture of the mandible and loss of several lower teeth.

In many instances, it is possible to manipulate the loose fragment of the upper jaw into proper position and immobilize it merely by wiring the teeth in occlusion. However, in some cases, forceful reduction of the maxillary fracture is entirely unsatisfactory and special appliances for reduction and immobilization become essential. To reduce a fracture of this type, I usually employ orthodontic molar bands, attaching one to the upper first molar tooth on each side. A jackscrew inserted between these two molar bands will force the loose fragment of the upper jaw outward (fig. 8*a*). Although such an appliance will push this fragment laterally, it will not correct the downward displacement. When the patient attempts to close his mouth, the teeth of this fragment come in contact with the teeth of the lower jaw, but an open bite is left on the opposite side of the mouth. To overcome this and force the loose fragment of bone upward into position, it is necessary to apply an arch bar to the unfractured portion of the upper jaw and a full arch bar to the teeth of the lower jaw.

Intermaxillary traction with rubber bands between these two arch bars (fig. 8*b*) will gradually close the open bite on the one side and, in turn, will force the loose fragment of bone on the opposite side upward into correct position (fig. 8*c*). Fixation of the fragments is maintained for four weeks.

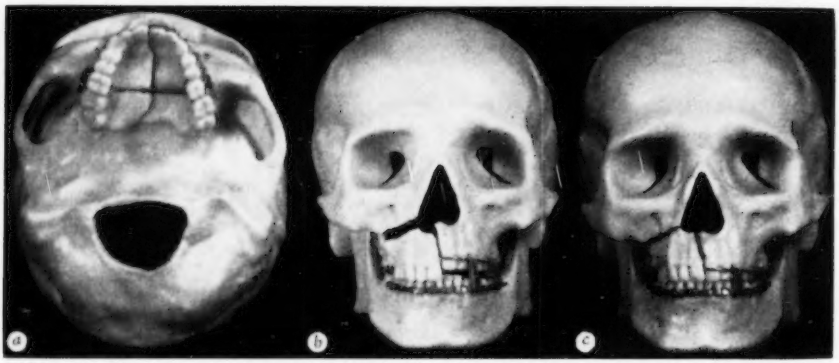

FIG. 8—(*a*) The loose upper fragment is forced and held outward by a jackscrew inserted between two molar bands as illustrated; (*b*) the downward displacement of the loose maxillary fragment is corrected by intermaxillary elastic traction on the opposite side of the mouth; (*c*) fracture reduced and teeth in occlusion.

SIMPLE FRACTURE OF THE UPPER ALVEOLAR PROCESS

Simple fracture of the upper alveolar process requires no special consideration in this paper. If the periosteum of the loose fragment of the upper alveolar process is not completely detached, the fragment should be retained and immobilized by a splint or intermaxillary wires. However, if the roots of the teeth in such a fragment are exposed in the line of fracture, it is, I believe, preferable to remove these teeth and even the fragment of bone if necessary.

Symposium: Facial Injuries

FRACTURES OF THE NASAL BONES

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THE nose, being the most prominent part of the face, is more frequently injured than many other portions of the body. Its close anatomic relationship to such structures as the vomer, cartilaginous septum and the ethmoid plate demands an understanding of the effect of trauma upon these structures and the resulting nasal physiology, since these structures are frequently more or less involved.

The various types of nasal fractures will be described under the following headings:

1. Simple fractures.
2. Compound comminuted fractures.
3. Nasal fractures in connection with multiple injuries of the facial bones.

Simple Fractures

The most common picture in this type of fracture is either displacement of one nasal bone inward or the entire nasal arch shifted to one side. In adults the ascending process of the maxilla is frequently involved in the fracture. Where the displacement of the bones is great, the diagnosis is obvious. However, when displacement is not severe and is masked by soft tissue swelling, one is likely to overlook the fracture, especially in children. A distinction should be made between fractures in children and those in adults. In the latter, the nasal bones are intimately fused in the midline, whereas in children they are separated. A common cause of fractures in children is a fall on the face, the impact of the blow usually being anteroposteriorly. The nasal arch may become separated at its base and the bones displaced over the ascending processes of the maxilla, resulting in considerable flattening of the bridge of the nose, the "open-book" type of fracture.

Compound Comminuted Fractures

In compound comminuted fractures the multiplicity of fractured bones causes a flatness of the bridge of the nose. In this type of fracture one often sees fracture or dislocation of the nasal septum with laceration of the mucous membrane and skin of the nose. The injury may be limited to the nose but often is associated with fractures of the other bones of the face, since the trauma is usually quite severe.

Examination and diagnosis. Diagnosis is made by the history of a blow and by examination. Swelling, deformity, abnormal motion or crepitus may be present. There may be epistaxis. Injury to the nasal mucosa causes impairment of breathing. Roentgenograms should be taken in all cases and are of much aid in doubtful cases.

Treatment. Relatively uncomplicated nasal fractures do not usually require hospitalization and the reduction may be accomplished in the emergency ward. Local anesthesia may be used for reduction of nasal fractures in adults but a short general anesthesia, such as nitrous oxide-oxygen, vinethene, or pentothal sodium, is preferred for children and nervous adults.

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A thin layer of moist cotton is wrapped tightly around the blunt end of a long periosteal elevator and then covered with petrolatum. The instrument is introduced into the nasal fossa, well under the depressed bone. The nasal arch is raised and the displaced bone is replaced with digital pressure. After the correction of the displaced fractured bone, I find that it is necessary to use an external splint to immobilize the fractured parts. This splint consists of dental compound moulded on the nose and held in place by adhesive plaster. If a splint is not used, especially in cases where the correction has been delayed a few days, the nasal arch will tend to revert to the displaced position. As in all fractures, early reduction gives a more satisfactory result. Late reduction often requires more strenuous manipulation of the fractured bones and the outcome is generally poorer.

Treatment of compound comminuted fractures. In comminuted fractures of the nasal bones, if treated while the bone fragments are still loose, the bones can be elevated and manipulated quite easily, but they are difficult to maintain in the desired position unless additional support is furnished. One of the easiest and most effective ways to control loose fragments of bone, is to pack the nasal cavity with gauze saturated with petrolatum. Intranasal splints are also effective in most cases. I prefer to use a *U*-shaped splint. This consists of a *U*-shaped wire, one arm of which is covered with dental composition and molded to the contour of the upper part of the nose. The *U* splint when in position is suspended to an external bar by elastic bands. I have seen house officers improvise a splint of this type from an ordinary large safety pin.

Nasal Fractures

A discussion of nasal fractures should not be limited to the bony framework alone. Any blow which causes a fracture and dislocation of the nasal bones if severe enough will also fracture and dislocate other important parts of the nasal framework and the nasal septum. It is often difficult to diagnose a dislocation of the cartilaginous part of the nose, but when the direction of force is against the tip of the nose and the columella, the septal cartilage may be forced upward alongside the perpendicular plate of the ethmoid. The cartilaginous septum may also be dislocated from its vomerian groove. Moreover, when the nose is struck by an "end-on" blow against the tip and columella, laceration of the mucosa below the edges of the nasal bones and backward dislocation of the lateral cartilages over the nasal bones usually takes place. Here again the treatment should be prompt before the soft tissues have a chance to repair themselves. The displaced cartilage should be replaced in its proper position. Septal hematoma, if present, should be drained promptly.

Complications

Simple fractures are not attended by complications as the healing powers are good in this area. However, if not treated properly, when the soft tissue reaction disappears, contour changes in the nose become apparent. In children, the defect is more glaring, and as they grow older the nose naturally becomes larger and the distortion of the component parts forming the nasal contour becomes more evident. Most of the distorted and twisted noses with marked deviation of the septum have their origin in childhood injuries to the nose. To prevent these deformities, when a child has a fall followed by nasal bleeding, a roentgenogram should be taken and if there is a nasal fracture, it should be treated immediately before the bones heal. The nasal septum should be carefully examined in all cases.

Infection may occur in severe compound comminuted fractures of the nasal bones. This may be prevented or minimized with adequate treatment and the use of antibiotics. Compound comminuted fractures may often be associated with laceration of mucosa and the skin of the nose. The treatment should consist of immobilizing the nasal bones and careful suturing of the soft tissues. Careful repair of the soft tissues will prevent stenosis and adhesions within the nasal cavity and will prevent conspicuous scars of the skin of the nose.

In severe facial injuries where there are fractures of other bones of the face, the nasal bones are invariably involved but the treatment of the nasal fractures is only a part of the general care which the patient receives. Such patients are usually gravely ill, therefore the fractures cannot always be treated in the same manner as simple fractures of the nasal bones. The main consideration of the operator is to give supportive treatment in order to avoid complications. One of the most serious complications that can occur in such cases is fracture of the cribriform plate and cerebrospinal leak to the nose. If nasal examination reveals escape of cerebrospinal fluid, the patient should be put in a semireclining position and instructions given not to blow the nose. Sedatives and the use of antibiotics are prescribed. Manipulative work within the nose should be avoided until the time when the cerebrospinal fluid loss stops. In my experience, this is usually within a few days, but if there is persistent leakage, consultation with a neurosurgeon is indicated.

INTERNAL FIXATION OF SEVERE JAW FRACTURES WITH STEEL PINS

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BY INVITATION

DIRECT, rigid fixation can be attained in severe jaw fractures by drilling one or more steel pins or Kirschner wires longitudinally through the bone across each fracture site to engage both fragments firmly. The resulting steel skeleton within the bone transforms a shattered, limp jaw into a solid unit and maintains its integrity until bony union has occurred and the pins can be removed.

Wide open operation is seldom desirable in jaw fractures because of blood supply problems and possible danger to branches of the facial nerve. Because of this, many distant and circuitous methods of fixation have been used in the past in which the bone fragments were attached indirectly to the opposite jaw, or to a plaster headcap, rather than to each other.

Wobbly fixation is no better in the jaws than in other fractures, and the end results may be even worse. The rigidity of the internal fixation described here is due to the fact that the steel pins have such leverage advantage that the bone ends cannot move unless they first shear the metal in two, and this they cannot do. The serious complications to be avoided in jaw fractures are osteomyelitis, nonunion and malunion; all of them can be prevented by early accurate reduction and firm fixation.

Fixation of mandibular fragments to the upper jaw by interdental wiring is still useful in simple fractures when the upper jaw is solid and when there are enough sound lower and upper teeth present on both sides of the fracture line. These prerequisites are not met in many fractures of the mandible, and when attempts are made to use this fixation without them, failures will occur. These failures will not be avoided by trying to extend the method with complicated, expensive, dental splints. Actually, the results may be worse if the jaw is left flopping for several days while such apparatus is being constructed and fitted.

Indirect fixation of the bones to a plaster headcap is cumbersome and insecure. The leverage is against the apparatus with every small movement of the headcap or intermediaries being translated into a larger movement of the bones. Even though it were possible to prevent the headcap from moving on the scalp, there is still no satisfactory method to prevent the scalp from moving on the skull. This floating fixation has resulted in many complications which could have been prevented by early direct fixation of the bone ends to each other.

Direct internal fixation of the bone ends to each other with steel pins or

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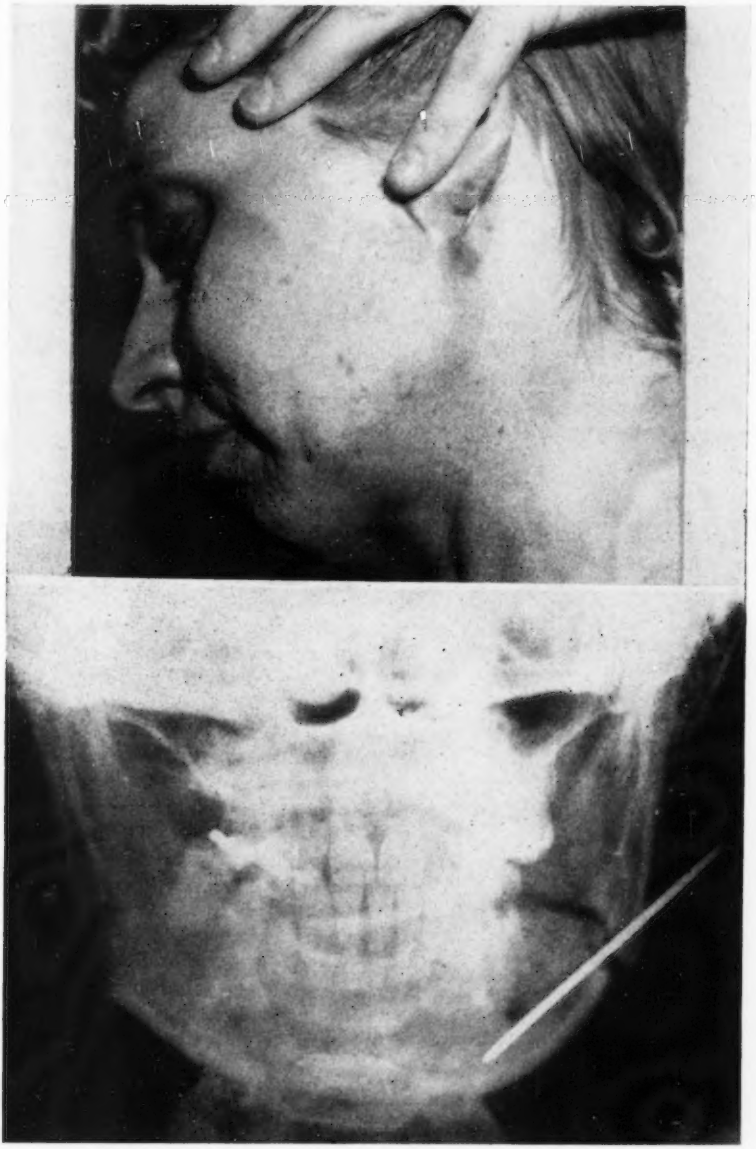

FIG. 1—Fixation of bad angle fracture, in which there was a tendency to marked displacement. The end of the pin is seen protruding behind the ear.

Kirschner wires has yielded good results in many severe fractures, even in atrophic, "pipestem," edentulous mandibles with multiple fractures that could not have been completely immobilized in any other way. The incidence of secondary bone grafting has been cut down almost to zero.

Since the method was published by us in 1942 for use in jaw fractures it has been widely adapted by others for use in other bones. This has confirmed its soundness, but the longest experience has been in treating jaw fractures. This experience has proved its dependability as the primary method to use in severe jaw fractures and has shown that with careful application it will yield the best possible results.

MANDIBULAR FRACTURES WITH EDENTULOUS FRAGMENTS

It is obvious that a complete fracture in an edentulous jaw is not a dental problem (except for later construction or adjustment of dentures) and is not amenable to dental fixation. The internal fixation described here is the best treatment for this type of fracture. In the same manner, internal fixation can be relied on for the successful treatment of fractures occurring in edentulous areas (e.g., behind the last tooth). Here, as elsewhere in the body, the fracture does better if both fragments are controlled and held in rigid apposition (figs. 1 and 2).

MANDIBULAR FRACTURES WITH PARTIALLY EDENTULOUS UPPER JAWS

When the upper jaw is completely edentulous, it is again obvious that interdental fixation cannot be used to control a fracture of the lower jaw. The same considerations hold when the part of the upper jaw opposite the teeth in the posterior fragment is edentulous, or when the upper or lower teeth in this area are broken or loosened by trauma or pyorrhea. In all of these situations, direct internal fixation of the bones is relied upon and any method of indirect dental fixation is uncertain or worse.

INTERNAL FIXATION OF FACIAL BONE FRACTURES

In many fractures of the middle third of the face or upper jaw, steel pins afford the best method of fixation. In unilateral fractures, while held in reduction, the pin is drilled through the loose side and then horizontally across through the sound side for cantilever fixation. In bilateral or crushed fractures, the pins may be used to hold the fragments together so that they can be immobilized further as a unit.

TECHNIC OF INTERNAL FIXATION

Under local or general anesthesia, the fracture is manually reduced and the two fragments held in apposition by the hands of one surgeon. The Kirschner wire is then drilled longitudinally down through the bone and across the fracture site by a second surgeon, using the electric drill for ease of penetration. It is important to aim the wire correctly as no adjustment of the fragments is possible after the wire is once in place. In general, it is best to keep the wire below the nerve canal and below any tooth roots. After the wire is in place, the projecting end is cut off about one or two centimeters outside the skin and is kept covered with a small dressing. The pin is removed in about six weeks except in very severe fractures, when fixation may be necessary for periods from eight to twelve weeks.

Standard operating room asepsis is observed and there has been no difficulty with infection. External dependent drainage may be done or omitted, according to the usual criteria. For fractures from the second molar area back

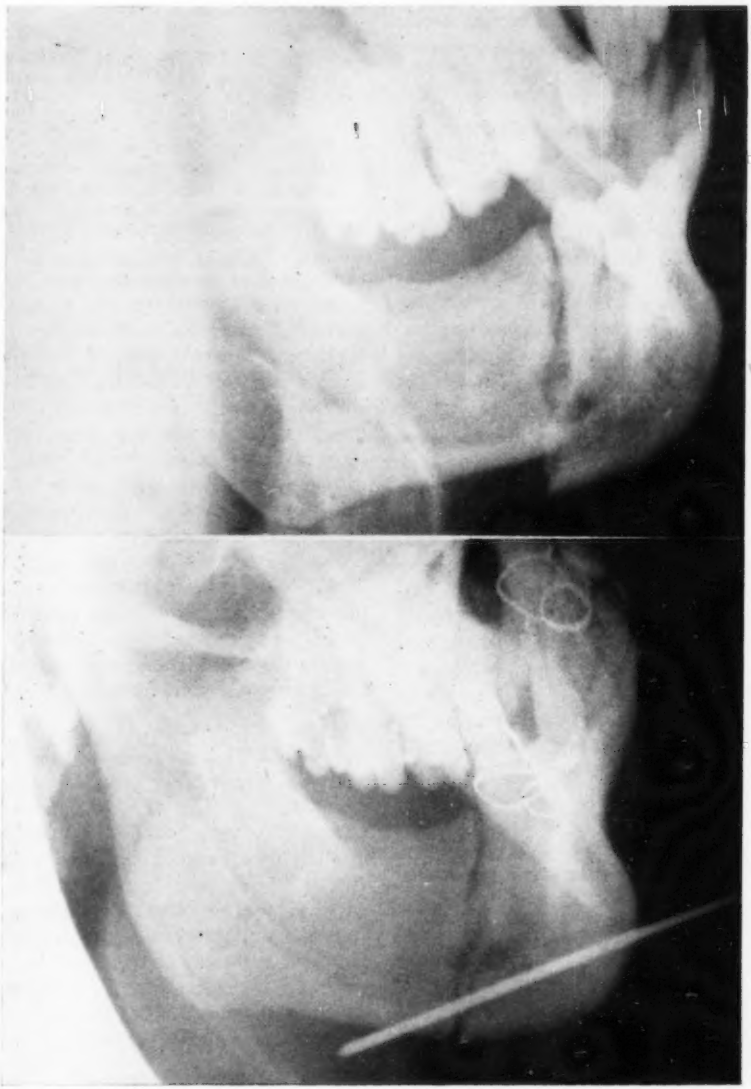

FIG. 2.—Reduction and fixation of long edentulous fragment. Interdental wiring used has auxiliary fixation for anterior fragment.

to the angle, it is usually best to drill the pin forward, starting from the posterior border of the ramus (fig. 1). For fractures anterior to the second molar region, it is usually best to start the pin anteriorly and drill backward (fig. 2).

The method is not especially easy, but it can be learned by almost anyone who has a good sense of direction, after a little experience with it. For the type of fractures for which it is used, it is probably the easiest, most direct, and most solid type of fixation.

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SINUS COMPLICATIONS OF FACIAL BONE INJURIES AND THEIR MANAGEMENT

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CRANIOFACIAL trauma frequently involves the nasal accessory sinuses. The degree of involvement is as variable as the variety and extent of the injury. It may be as simple as a localized fracture of the canine fossa or a depressed fracture of the external table of the frontal bone, or it may be as massive as a crushing compound fracture of the skull and face with direct trauma to the brain. The causes are equally varied, embracing penetrating wounds by missiles, fractures due to blows with various instruments, and falls from a height or from moving vehicles. In view of the multiplicity of etiologic factors and the extreme variety of degree and extent of the injury, a limited presentation such as this can hope to cover only the basic problems involved.

The importance of the role of the sinuses in these conditions can be fully appreciated only if one bears in mind the fact that the lining membrane of the sinuses, being continuous with that of the nasal cavities proper, offers a potential source of infection which is more threatening than a laceration through the soft tissues. The reason is obvious. A compound fracture through the overlying skin and soft tissues is open to view from the very beginning and can be promptly evaluated and dealt with, whereas if the trauma involves the skull and the sinuses without rupture of the soft tissues, the potential source of infection is in the depths and its possibilities are unknown.

The incidence of sinus involvement depends on the type of trauma under consideration. Statistics are available only in certain classes of injury. Alexander,[1] for instance, states that in a series of 421 cases of war trauma to the face there were 56 in which the sinuses were involved, 42 affecting the maxillary, 10 the frontal, and 4 the ethmoid. Fehr[4] studied 417 cases of basal skull fracture in which 4.8 per cent developed meningitis from involvement of the sinuses.

The maxillary sinus is affected in practically all fractures of the maxilla, particularly those in which the orbital floor is involved. There is more or less laceration of the lining mucosa, causing blood to accumulate in the sinus. Also, frequently foreign bodies and bone fragments are present. Roentgenograms in almost every case will reveal a clouding of the sinus. The indications in such cases are the removal of foreign bodies and loose bone fragments, and the provision for drainage via the inferior meatus. This can be readily accomplished at the time the fracture is being aligned. In most cases both objectives can be attained through a Caldwell-Luc exposure. In those cases where the soft

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tissues have not been lacerated and repositioning is contemplated by means of wiring or intraoral appliances, it may be advisable to provide drainage via an intranasal antrostomy. In no instance should one resort to irrigation. Blood clots can readily be removed by aspiration.

It should be emphasized that bony fragments which are still attached to the soft tissues should not be removed, since in most cases they can be salvaged. With the routine employment of antibiotics, infection rarely occurs and osteomyelitis is extremely uncommon.

Fractures involving the frontal and ethmoid sinuses are much more serious than those affecting the antrum, because of their close proximity to the endocranium. A break in the continuity of the roof of the ethmoid labyrinth, the cribriform plate or the posterior wall of the frontal sinus, provides a direct pathway for infection, and since the dura is then the only barrier to a spread to meninges and cerebrum it is of utmost importance to ascertain whether a laceration is present or not. The appearance of a rhinorrhea or a subdural or intracerebral aerocele makes the diagnosis certain. There are some cases, however, in which the dura has been lacerated and yet there has been no rhinorrhea or aerocele. This will happen occasionally when intracerebral pressure forces the anterior pole of the frontal lobe into the laceration, thus sealing it off. It is extremely important that the diagnosis of a laceration of the dura be made whether rhinorrhea or aerocele is present or not, because if left unrepaired, even though primary healing is obtained, a subsequent upper respiratory infection may lead to a fulminating meningitis. It was pointed out by Calvert,[2] who reviewed 103 war injuries involving the frontal and ethmoid bones, that the diagnosis of a dural laceration in the absence of a rhinorrhea could be assumed under the following circumstances:

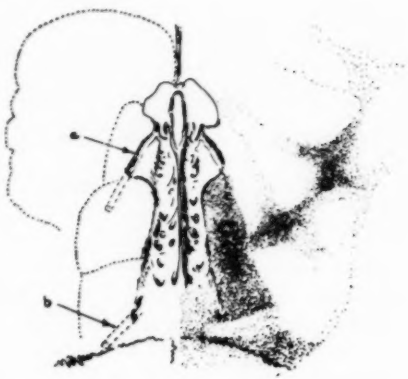

FIG. 1.—Roof of ethmoid. (a) Anterior ethmoidal vessels; (b) posterior ethmoidal vessels. Dotted lines show outline of ethmoid cells and orbit. (C) Dense layer of roof of ethmoid; (D) dense areas of roof of orbit (from Johnson and Dutt[5]).

1. Roentgenograms showing a wide gap in the posterior wall of the frontal bone or any marked displacement of bone in that area.

2. A fracture across the base from one anterior fossa to the opposite.

3. Projection of a sharp fragment of the posterior wall posteriorly.

4. A fracture line that widens as it descends the posterior wall into the ethmoid sinuses.

5. Displacement of the frontozygomatic synostosis when associated with tilting of the crista galli.

Naturally, in all cases much will depend on the type of roentgenograms taken and their interpretation. In many cases it is extremely difficult to demonstrate a fracture line in the vicinity of the cribriform plate and the ethmoid roof because the line is frequently lost in the mass of overlapping ethmoid cells. Johnson and Dutt,[5] who had experience with 98 cases of brain injury involving the sinuses, showed a number

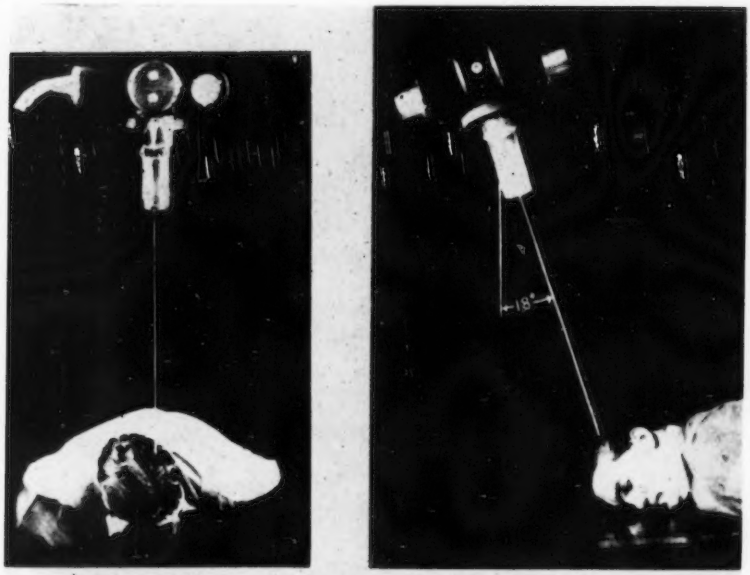

FIG. 2—Tube and head position for the oblique view of the ethmoids[5] (projection A). The tube-film distance is 36 inches. (A) Sagittal axis of head 45 degrees inclination to table; central ray through center of orbit. (B) Tube inclination 18 degrees; central ray through nasion. This projection is recommended by Johnson and Dutt[5] to show the roof of the ethmoid labyrinth.

of illustrations depicting the vagaries of fracture lines which were demonstrated by their modification of a projection attributed to Graham Hodgson. It is an oblique view in which the ethmoids and frontals are projected through the orbit, showing the openings of both anterior and posterior ethmoid vessels, the suture line between the orbital plate of the frontal and the lamina papyracea and an unobstructed view of the orbital roof (figs. 1, 2 and 3).

The question then arises as to what should be done and when. It is generally agreed that as soon as possible after the injury, after shock and hemorrhage have been controlled, the patient should be taken to a center where skilled attendants with adequate equipment are available. As soon as the above requirements have been fulfilled and the diagnosis has been made, the indications are to repair the damage and seal off the endocranium. This requires adequate exposure, thorough cleansing, removal of foreign material and devitalized tissue, realignment of displaced bone and closure of the dural defect.

It must be borne in mind that there is a difference between a fracture into the sinuses without laceration of the soft tissues and an injury due to penetration of a missile or foreign body. As a rule penetrating wounds call for immediate therapy, even when a thorough analysis of the extent of the injury is still incomplete, because the wound tract is infected and must be cleansed and repaired. Treatment of trauma due to blunt violence without penetration by missiles or foreign bodies usually can be deferred long enough to permit

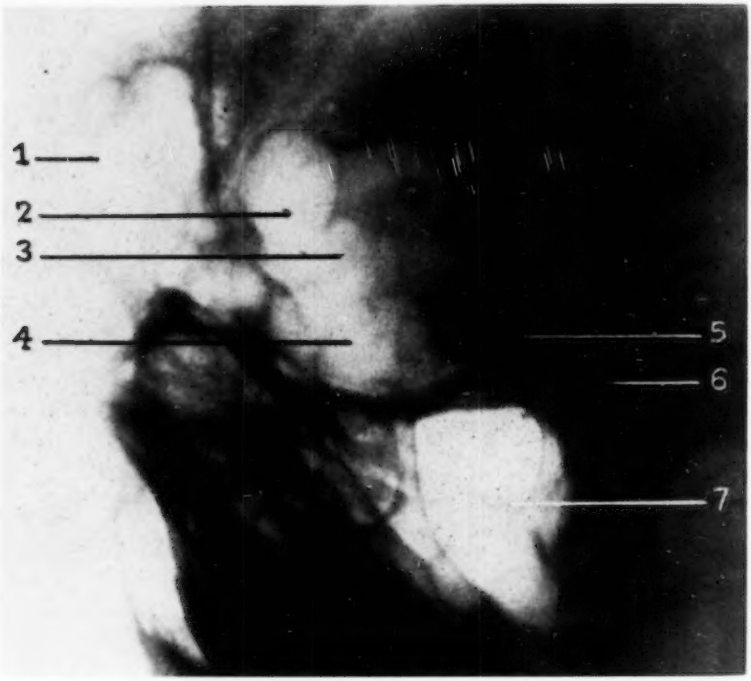

FIG. 3—Typical picture from projection as shown in figure 2. (1) Frontal sinus, opposite side; (2) frontal sinus, same side; (3) orbital extension of frontal sinus; (4) ethmoid cells; (5) optic groove; (6) optic foramen; (7) sphenoid sinus projected through antrum, same side.

transportation to a center where the facilities are adequate and a thorough appraisal of the extent of the damage can be had. Naturally, antibiotics are given from the start and add a considerable measure of safety, particularly if there is a delay in instituting adequate surgical procedures.

Dural lacerations may be closed either by direct suture or by fascia lata or muscle flaps. The question as to whether the intradural approach is preferable to the extradural is one that has to be decided according to the location of the laceration and the extent of the trauma. Most brain surgeons prefer the intradural approach because it affords a better exposure of the floor of the anterior fossa and the structures in the post-ethmoid, sphenoid and pituitary area. On the other hand, the approach through the frontal sinuses provides a better visualization of the sinuses themselves, which frequently must be exenterated or drained to prevent infection. In general it may be stated that the extradural approach is preferable if the dural-sinus defect is anterior to the groove for the anterior ethmoid vessels, while the intradural approach is recommended when the defect is located posterior to the groove.

What to do with the sinuses is another matter. It depends on the type and extent of the injury as well as the size and extent of the sinuses. The prime considerations here are to eliminate factors which might predispose to secon-

dary or late infection, restore the function of the sinus if possible and, lastly, to repair the cosmetic defect. Restoring the function implies the re-establishment of the sinus with adequate drainage into the nasal chamber and the elimination of obstructions and blind pockets. Where the loss of bone is considerable this may be impossible of attainment, and it may be necessary to obliterate the sinus. In fact, if the frontal sinus is small, it is advisable to obliterate it in any case since the resulting deformity would be slight, and by bringing the skin into contact with the dura the latter can be effectively sealed off. If the ethmoids are uninvolved, they should be left alone. If infected or if a fracture extends through them into the cribriform area, they should be exenterated. In the case of a large supraorbital cell, enough of its floor should be removed to afford adequate drainage into the ethmoid labyrinth which in such a case would have to be exenterated. If the frontal sinus is deep or extensive laterally, wide drainage should be provided into the nose, and in such cases it is necessary to remove the ethmoid cells. The prime objective in every case is to see that no pocket or air-containing cavity is left behind which is not provided with a free drainage channel into the nose. In case of doubt it is always safer to obliterate the sinus despite the deformity, which can later be corrected by a plastic procedure.

Despite the extensive trauma sustained in many of these cases, it is agreeably surprising to note the high percentage that recover. Calvert[3] reported 57 cases with only 4 deaths; Johnson and Dutt,[5] 20 cases with 7 deaths; Lumsden,[6] 20 cases with 1 death.

The importance of diagnosing and repairing dural defects is strikingly illustrated in Calvert's report of 43 cases, 20 of which were treated conservatively and only 7 recovered, whereas of the 23 treated by dural repair 20 recovered. It is therefore apparent that expectant treatment with reliance on antibiotics alone will not cure the majority of patients.

Before closing I should like to call attention to a group of cases in which a foreign body has been projected into the sinuses without damage to the bony walls separating them from the endocranium and without damage to the latter. In many instances the point of entry is nowhere near the final resting place of the foreign body and in some cases the latter is fragmented by the impact, thus resulting in more than one foreign body. If the foreign body is opaque, it can be located by careful roentgenographic studies. However, in some instances the foreign body fails to show in the roentgenograms. Objects of this character, however, are irritating enough to produce a draining fistula which is evidence enough of their presence, and this should therefore be an indication for an exploratory operation.

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SOFT TISSUE WOUND REPAIR

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SINCE this paper is a part of the symposium, it will deal only with the diagnosis and treatment of fresh wounds of the face. No attempt will be made to discuss late wound problems and their methods of repair.

Remembering that in any treatment of traumatic wounds the entire body is usually involved and that this paper pertains only to one part, the primary necessities of the surgical shock must first be considered. Assuming that these essentials have been attended to and that the patient is in good condition, then and only then the problem with which we are concerned, namely, the early primary closure of deep soft tissue wounds about the cheek, may be considered.

Since there is usually much ecchymosis, tumescence, active bleeding and distortion of facial features, stereoscopic roentgenograms of the facial bones are taken in order to determine the extent of the trauma and the presence or absence of fractures. If fractures are present, these are usually repaired at the same time as the early primary closure of deep soft tissue wounds.

As a rule, soft tissue wounds may be closed by primary suture within twenty-four hours. In treating early deep lacerations about the face, accurate diagnosis of the parts injured is a primary consideration. To overlook a lacerated parotid duct or injured peripheral facial nerve can be most disastrous. A Cameron headlight and magnifying loupe eliminates guesswork in diagnosis and is of great value in the repair of severed branches of the facial nerve.

In most cases of lacerations without fractures, local anesthesia is sufficient. However, the primary concern is adequate careful cleansing. All foreign bodies should be removed to prevent suppuration and their unsightly appearance in a healed wound, as they usually are visible through the scar. Irrigation usually removes the larger particles, but smaller ones are manually removed with small pointed forceps. The skin and skin edges are cleansed with soap solution while the wound is adequately irrigated with warm sterile normal saline.

All hemorrhage should be controlled meticulously either by clamping or with fine 4-0 white silk sutures. Blood clots are removed, usually by irrigation. Very little debridement is necessary in facial wounds as the copious blood supply prevents devitalization. If the wound is jagged and irregular, the skin edges can be sparingly trimmed.

Deep lacerations about the cheek involving the branches of the facial nerve must be examined carefully. Usually a paralysis can easily be noted, but a functional examination must be made to determine the extent. After this a search must be made for the severed ends. This in most cases is tedious and difficult as the peripheral branches are exceedingly small. If one is fortunate enough to find a larger branch, this can be anastomosed with 6-0 or 7-0 silk. A sterile faradic

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stimulator and magnifying loupe help considerably in this search. The smaller branches which defy anastomosis should be brought together as close as possible by suturing fascia and muscle. Fortunately most of the paralysis usually seems to disappear when the wound is healed even though direct anastomosis of the smaller branches is impossible.

All deep wounds should be closely examined to see if the parotid gland or duct has been lacerated or severed. If the gland itself has been injured, the capsule should be sutured tightly with interrupted silk and the wound closed without drainage. If the duct has been severed, pressure on the gland will usually bring a flow of secretion from the proximal end. Again the magnifying loupe tends to make the search easier. Since there has been a very high number of fistulas from severed ducts, many methods have been devised for their repair. To date there is still no universal agreement as to the correct procedure. If the duct has been severed cleanly and there is a chance to anastomose, this is the method of choice. A tantalum or silver probe is usually inserted into the wound through the proximal end and the distal end, and 6-0 silk is used in interrupted sutures. Whether to leave the probe in place has not been settled as yet. The majority of opinions seem to favor its removal. If the injury has occurred in the masseter portion of the duct, where it is better developed, there is a much higher rate of successful anastomosis. When the injury has occurred nearer to the gland proper, where the wall of the duct is much thinner and more friable, the incidence of fistulas rises. In searching through the literature an excellent article by Dr. Sparkman[1] of Dallas brings to one's attention that a very simple method described during World War I has been forgotten; namely, the method of Morestin employing the principle of ligation of the proximal end of the duct, thus bringing about a physiologic cessation of gland function with accompanying atrophy. This seems to be the best method of approach to the problem of the duct when it is injured near the gland. Many other methods have been tried. One worthy of mention is that of applying a seton from the region of the proximal end of the duct into the buccal mucosa and allowing a tract to form into the mouth. The external wound is closed tightly in these cases.

All primary repair should be in layers. First, the deep muscles should be repaired. 4-0 silk can be used. Then the superficial muscles of expression should be approximated. All dead spaces must be obliterated. The skin edges should be undermined to make the resulting scar more inconspicuous. 4-0 silk with atraumatic needle is used to approximate the skin edges. The sutures are placed close together and tied without tension. A vaselin gauze strip is placed over the wound. This prevents the formation of a hard collection of dried serum which tends to separate the superficial skin edges. Gauze dressings are then applied. Cotton mechanics waste is then placed over the wound and held in place with an ace bandage which is carefully applied and anchored with cross strips of adhesive.

Sutures are removed early, half on the first postoperative day and the remainder on the following day. If the wound edges have any tendency to separate, elastoplast strips are placed at right angles to minimize the scar.

In conclusion, the principles involved in the repair of soft tissue wounds involving peripheral facial nerves and parotid gland and duct lesions have been presented. Special stress has been placed on accurate diagnosis and repair, using the magnifying loupe. I am certain there

are many other methods, but in general if these principles are followed, lacerations involving the cheek will be handled successfully.

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Symposium: Facial Injuries

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DIAGNOSIS AND SURGICAL TREATMENT OF FRACTURES OF THE ORBIT

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DIAGNOSIS

DIAGNOSIS of fracture or dislocation of the orbital bones is made by a detailed history, which includes time of injury and type of object traumatizing the orbit, and a careful and thorough study of good roentgenograms in the Caldwell and Waters positions, showing the entire orbits and the superior maxillas. Because of swelling which is frequently present, physical examination, including inspection and palpation of the orbital region, may be misleading. Also, I would emphasize the careful study and observation of any diplopia in all cardinal directions of gaze. It has been helpful to observe straight upward and straight downward movements of the eye, which give a good indication of the limitation of the movement of the injured eye.

The cover test is essential for the detection of phorias or tropias, but should be combined with a statement from the patient that such a condition had not previously existed. We feel that the positive diagnosis of fracture, associated with more than a few diopters of diplopia, justifies or forces one to recommend surgical treatment, whether it is a simple fracture or combined with displacement of the floor or the rim of the orbit.

If a patient with a recent injury of the orbit shows any diplopia and/or enophthalmos, the case should be considered a fracture dislocation of the orbit until proved otherwise. Downward displacement of the floor of the orbit into the maxillary sinus is frequently hard to demonstrate with a roentgenogram in the Waters position because, first, the floor of the orbit is so thin that it usually does not show a shadow of displacement. Second, there is usually a marked increase in density of the maxillary sinus, in recent cases, from a blood clot which may fill, partly or entirely, the maxillary sinus. Therefore, this marked increased density of the maxillary sinus frequently obstructs the view of the fractured and dislocated floor of the orbit into the maxillary sinus. Lateral roentgenograms are seldom

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The slides were prepared by Miss Jean Robinson and Mrs. B. F. Woodford of the Memphis Optical Dispensary.

helpful in diagnosing fractures or dislocations of the floor and inferior rim of the orbit.

While trying to make a diagnosis of orbital fracture the patient should be observed daily for the first week after the accident and every other day for the second week until a conclusion can be reached, whether the injury is of such nature that it should be treated surgically or nonsurgically. If a patient is in shock from the accident or the roentgenograms cannot be made adequately, wait until the patient is well enough to be moved and placed in the proper position for them to be made. A poor roentgenogram may be worse than none and can lead to an error in diagnosis.

FIG. 1—Shea-Anthony intramaxillary sinus pressure balloon. (Photograph by James A. Lawrence.)

TREATMENT

There are two types of treatment; (1) for recent fractures and (2) for healed fractures of the orbit.

Recent Fractures

The diagnosis and treatment of recent fractures has been covered frequently in the literature but, unfortunately, has not been described adequately. If a fracture or dislocation of some part of the orbit is causing enough trouble to produce marked diplopia, marked restriction of movement of the eyeball with enophthalmos, or backward and downward displacement of the rim of the orbit, it is our opinion that superficial adjustments are uncertain and frequently insufficient to correct functional and cosmetic defects.

If there is a fracture of the orbit with no displacement, with slight diplopia, slightly restricted movement of the eyeball in any direction, no enophthalmos, and only slight exophthalmos from swelling of the contents of the orbit, occasional fracture dislocations in the inferior rim of the orbit may need no surgical treatment. In a case of recent injury where there is doubt whether to operate or not to operate, it is safest to operate because you will not make the patient any worse and you probably can remove a slight cosmetic and functional disability that cannot be corrected satisfactorily later with surgery.

For several years I have found that recent fractures of the orbit can be most satisfactorily corrected cosmetically and functionally by making a slit horizontal antrostomy anteriorly in the inferior meatus close under the surgically fractured inferior turbinate. The reason we make the antrostomy of this shape is to keep the inflated balloon from pressing on the inferior turbinate and septum. A completely deflated Shea-Anthony intramaxillary sinus, water pressure, Latex rubber balloon* (fig. 1), oblong in shape, which carries a 12 inch stopper stem, is inserted. After it is inserted in the maxillary sinus, it is filled with water to create a marked pressure sufficient to displace the fractured dislocated bones of the orbit beyond the normal position, which sometimes requires one

* Made by C. R. Bard, Inc., Summit, N. J.

to five minutes constant pressure from the balloon. When a difference in the palpation is noted by careful observation before and after pressure is applied, and if too much swelling is not present, you may reduce the pressure just enough to maintain the fractured dislocated bones near their normal position.

To reduce a fracture of the rim and/or floor of the orbit, I have used as much as 30 cc. in this balloon. The average male adult usually needs about 15 cc. to 20 cc. of water and the average female adult, 10 cc. to 15 cc. Be sure to use a marked amount of slowly increased pressure for reducing the fracture and a medium amount of pressure to maintain a normal position of the fracture. It is usually best to do this operation under local anesthesia with moderate sedation as the patient can frequently tell you how much pressure is being created by the way it feels in the maxillary sinus. While the fracture is being reduced, the patient will have a moderate or considerable amount of pain in the region of the maxillary sinus, and when pressure is reduced, the patient will feel a soreness or fullness for twelve to twenty-four hours. After that, there will be little discomfort.

After the operation, you may x-ray the patient again if you care to check on the reduction. Usually you will get a good picture because the rubber balloon does not interfere with the density of the picture enough to destroy the value of the postoperative diagnostic roentgenogram. Never fill the rubber balloon with opaque oil because it interferes with the postoperative roentgenogram reading and it causes a deterioration of the balloon. If one finds it necessary to use an opaque solution for some postoperative roentgenogram readings, a mixture of 20 per cent barium sulfate and 5 per cent acacia mixed with water may be used. This mixture is cheap and does not cause deterioration of the balloon. The balloon can be used more than once if properly cared for.

The Shea-Anthony high pressure balloon is smaller with a much thicker wall and longer stem than previous balloons made for this purpose. The thick balloon will stand more pressure; the stem will not blow out; it will not be punctured as easily by the fractured bones; it will not cause as much intranasal pressure as the previous thin balloons which tend to crawl into the nose.

If possible, we like the intranasal antrostomy operation in recent fractures of the orbit best because it prevents surgical destruction of the necessary nerve and blood supply to a few teeth, perhaps three or four. Dr. D. B. Parker says: "The teeth, having an almost terminal circulation, have no adequate collateral circulation. When trauma injures the hair-like apical artery entering the root tips to supply circulation to the dental pulp, it renders the tooth particularly susceptible to infection involving the pulp because regeneration of that tissue cannot normally take place. When the circulation is destroyed, the result is a decomposition of the pulp which cannot readily be carried away as a waste product, and apical infection by organisms carried through the blood stream involves the periapical tissues."

If, for some unknown reason, you cannot reduce the fracture as described above with a pressure balloon, make the same antrostomy described above and make a sufficiently small high opening in the canine fossa to reduce the fracture by pressure with blunt instruments or by packing gauze tightly in the antrum. Then insert the balloon in the same way described above. I try to make the canine fossa opening as small and as high as possible, to prevent injury to nerves and blood vessels or teeth.

We have been using an Anthony and Fisher intramaxillary sinus orbital floor

FIG. 2.—Anthony and Fisher intramaxillary sinus orbital floor and rim reducing fracture forceps. (Photograph by James A. Lawrence.)

and rim home-made reducing fracture forceps* (fig. 2). This forceps opens when the handles are compressed. The outside surface on one side of this forceps is checkered to give it a rough, nonskid surface. This is used principally for reducing the rim of the orbit. The other side has a pivoting, flat, rounded surface with a knob attached to the end of the forceps to reduce the very thin floor of the fractured orbit by placing it against the mucous membrane of the intramaxillary sinus, instead of using pressure against gauze packing. We think this intramaxillary sinus reducing forceps causes less trauma to the mucous membrane and floor of the orbit than other methods and instruments and the amount of force can be perfectly controlled.

The soft tissues of the canine fossa are closed tightly with interrupted catgut sutures and the stem of the balloon is brought out through the intranasal antrostomy described above. The stem is fastened loosely over the ear with adhesive. These pressure splint water balloons are left in the maxillary sinus from one to three weeks, the average duration being two weeks as the bones of the orbit heal very rapidly because of generous blood supply and local immunity of tissues.

After the balloon has been in the maxillary sinus a sufficient time, deflate the balloon very slowly and gently make traction on the stem and the balloon will slip through the intranasal antrostomy. Do not irrigate the maxillary sinus for one week after the balloon is removed.

I have never had an infection from using the above method, but I have always used penicillin or sulfonamides before, and for a few days after, the operation to discourage infection.

Usually diplopia is relieved or improved immediately after operation and will greatly improve from one to six months after operation. Do not expect any improvement after six months, but a little remaining diplopia is not usually disturbing to the patient because it is only detected when the patient looks extremely up and extremely up and out, and this is not a necessary direction of gaze. He soon learns to protect himself from this slight diplopia.

I feel sure that this method of treatment gives far better results than packing the maxillary sinus with tight gauze, which has been recommended by so many men for a long time. However, we think packing the antrum very tightly with gauze is the next best treatment.

In severe recent fractures of the orbit, there usually is no fracture of the malar bone but a fracture at the attachment of the malar bones with the adjacent bones. The malar bone is much thicker, and the weak point is its attachment to other bones. The floor of the orbit fractures and dislocates easily

* Made by Ewing Cannon, Memphis, Tenn.

downward into the maxillary sinus because on the mesial side of the inferior orbital canal the bone is usually about 1 mm. thick, and on the temporal side of the inferior orbital canal the bone is about 2 mm. thick. Considerable or slight fracture dislocation of the floor of the orbit (orbital plate of the maxilla) may occur from a direct blow on the eyeball only without causing damage to the eyeball or the bony rim of the orbit. However, intraocular lesions of the anterior segment of the eye or the fundi, such as optic atrophy, a dilated pupil or a ruptured choroid, not uncommonly occur from severe contusion of the eyeball. Therefore, a careful vision test, field test and ophthalmoscopic examination of the fundi, after the pupil has been dilated with a mydriatic agent, should be made as soon as possible after the accident. Ocular lesions should be treated as indicated.

When there is a complete laceration of the lower canaliculus associated with fracture of the orbital bones, one can inject sterile milk into the upper canaliculus and the severed nasal end of the lower canaliculus can be identified for repairing.

Healed Fractures

Healed fractures of the orbit, two weeks or longer after the accident, can be reduced by making a small high incision through the canine fossa, reducing the floor of the orbit as described in "recent fractures," and/or cutting the dislocated rim of the orbit vertically in two places with a very small chisel through the canine incision, and inserting the pressure balloon to hold the fractures in place from three to four weeks. Old healed fractures (of months or years duration) of the floor and/or rim of the orbit may cause a mucocele of the maxillary sinus. This constant pressure of mucus in the intramaxillary sinus may be sufficient to cause atrophy of the very thin bony floor of the orbit. If you think the balloon will not hold the severe fractures satisfactorily, or if there is a bony absence of floor and/or rim of the orbit, a rigid, permanent, stationary, intramaxillary sinus jackscrew (fig. 3) or cantilever jack, made of SMO 316 stainless steel, can be inserted in the antrum. This carries an orbital plate which can be adjusted to hold the floor and/or rim of the orbit up while it heals. Always make a large, permanent, intranasal antrostomy during this operation for permanent drainage following operation. We have operated on a few cases using this type of jack, and the results have been satisfactory. This metal, permanent type of jack has not caused any discomfort, no increased drainage from the antrum, no pressure necrosis in the bones, for the jack has no spring in it and makes no great pressure but just holds the bones up in a normal, or near normal, position. As far as I know, our intramaxillary sinus, rigid, stainless steel jack has never been advocated before. These jackscrews have been very satisfactory, but we have found that they are too expensive and too difficult to manufacture, so we have changed the type of jacks to a stainless steel SMO 316 adjustable tube jack which is made in various lengths and the use of these will be reported at a later date. I do not

FIG. 3—Anthony and Fisher intramaxillary sinus stainless steel jackscrew.

advocate the implanting of bone, cartilage or anything else in the orbit to correct a recent or healed fracture of the orbit causing distressing diplopia or enophthalmos. The diplopia is either due to dislocation of the globe downward and/or incarceration of the inferior rectus muscle. The proper way to correct this is to push the entire contents of the orbit up into normal position so that all the extraocular muscles can function. Inserting a substance in the floor of the orbit too often interferes with the normal action of the inferior rectus muscle and the inferior oblique muscle. Bone and cartilage have a tendency to absorb with time. Other foreign substances usually migrate to a new position on the floor of the orbit, cause chemical irritation, expel themselves, or have to be removed. It is also impossible to control the amount of correction.

It is primarily the responsibility of the otolaryngologist, the ophthalmologist and the plastic surgeon to make a correct diagnosis and decide whether the patient should be surgically or nonsurgically treated, because he is concerned with functional examinations.

In a recent or late fracture, if you cannot keep the rim of the orbit in position with the above method alone, then it is indicated, by open reduction, to wire the two fragments together with very fine (no. 32) stainless steel wire and leave the wire in permanently. At the same time, you should use the balloon or jack for the fractured floor of the orbit.

If there is a traumatic complete unilateral or bilateral fracture of the superior maxilla, associated with downward or backward dislocation, then it is good surgery to wire the teeth together in their normal occlusion and use the above methods for the rim and/or the floor of the orbit. Wiring the teeth together will help to hold the orbit in its normal position after Adams vertical internal wire fixation procedure has been accomplished. (Use no. 24 stainless steel wire.)

The operator who takes the responsibility of diagnosing and treating the orbit should equip himself with special instruments and supplies and have his various plans of attack thought out before the injury occurs.

Since the capacity of the maxillary sinus is 10 cc. to 20 cc. and the average capacity for an adult is 15 cc., this gives the balloon ample room for expansion with water pressure. Complete traumatic lacrimal canal obstruction is best corrected by dacryocystectomy.

When there is a severe complicated injury, do not hesitate to call in the ophthalmologist, the otolaryngologist, the plastic surgeon or the oral surgeon for consultation, for, in some cases, there is a place for one or all of these physicians. The magnitude of some of these cases thoroughly justifies the expense involved in employing more than one surgical specialist.

The majority of orbital fractures occur from automobile accidents associated with injury to other parts of the body. Fights, sports and mechanized machinery are next in order, causing these fractures. Therefore, we can expect an increase of fractures of the orbit over the number that have occurred in the last quarter of a century.

Healed or recent orbital floor and/or inferior rim fractures of children are a unique and difficult problem in both diagnosis and surgery. No satisfactory diagnosis and surgical correction has been reported. The density of the bone is low, making some fractures difficult to visualize with roentgenograms, and extraocular muscle function tests are done with poor cooperation on the part of the patient. All permanent teeth do not

erupt until a child is about 12 years of age, except the third molar, and opening the anterior wall of the maxillary sinus before this time may destroy permanent teeth in this region. Therefore, a very high inferior meatus intranasal antrostomy and use of the pressure balloon in the first week is much more important on children than adults. The procedure is much more difficult, for the nose is small and the inferior or low middle meatus space is difficult to work in. Dr. Adrian J. Delaney and Dr. Harry R. Morse say: "The maxillary sinus enlarges with the development of the facial bones and the tooth buds. At birth, the antrum is at the level of the upper border of the inferior meatus, and during the course of the next few years, assumes a close relationship with the inferior meatal wall. At the age of 15 years, the antral floor is lower than the nasal floor."

The Shea-Anthony balloon is usually too large for small children and one must use a small balloon, such as the Robinson intranasal maxillary sinus balloon, or the size 12 or 16 French catheter retention balloon* which has 5 cc. or 30 cc. capacity. An ice pick may be of value to elevate a depressed rim.

Healed fractures (over one week) are a more serious problem and, unless there is a marked diplopia, enophthalmos and restricted movement of the eyeball, the case should be delayed for months or years until the permanent teeth have erupted. The severity of each individual case must help the physician to determine whether the teeth should be sacrificed for correction of orbital fracture before eruption of all the anterior permanent teeth. The Anthony-Fisher orbital fracture reduction forceps will allow mobilization in children through a smaller opening in the anterior antrum wall than the use of ordinary instruments for reducing fracture.

* Made by C. R. Bard, Inc., Summit, N. J.

CONCLUSION

For orbital fractures, I find surgery with the intramaxillary pressure balloon gives much better functional and cosmetic results than any other treatment. The operation is simple in comparison with other surgery to correct orbital fractures and the instruments and balloons used in this operation are few and inexpensive. The majority of early cases can be reduced and reduction can be maintained with the intramaxillary high pressure balloon. Thus, a canine fossa incision is often omitted and may prevent devitalizing some of the teeth by the operating procedure. Last, but not least, many patients will let you do the intranasal antrostomy intramaxillary sinus pressure balloon operation early when they will not consent to early operation through the canine fossa. With this balloon operation, the surgeon will consent to operate on more and earlier cases and, in turn, he will have a better percentage of good results in cases of a severely injured orbit.

A displaced orbital floor fracture creates space for the orbital contents, the greatest space being in the region under the posterior half of the globe.

The amount of enophthalmos and/or diplopia present following an old healed depressed fracture displacement of the floor of the orbit does not always indicate the extent of the fracture displacement of the bony floor of the orbit.

Orbital fractures, on some occasions, may cause fracture displacement with traumatic closure of the lacrimal duct. Early replacement of the fracture may save a permanent occlusion of the lacrimal passage.

The best rubber balloon connector is made by Becton & Dickinson, no. 470, to be used with a Luer-Loc control syringe, size 3, 5 or 10 cc. With this connector and syringe, one has better control and can estimate how much pressure is being put in the pressure balloon.

Restoration of functional result is of primary importance. However, cosmetic result should be considered as well and will create inspiration for better functional result.

Fractures of the orbit in very young children should be given more consideration than has been given to them in the past.

ACKNOWLEDGMENT

I wish to thank Daniel F. Fisher, M.D., for the use of his excellent material in helping to prepare this part of the symposium on "Facial Injuries."

ELECTRONIC FLASH ATTACHMENT FOR CLINICAL PHOTOGRAPHY

ALBERT H. ANDREWS, JR., M.D.
CHICAGO, ILL.

ELECTRONIC flash equipment has been adapted to clinical photography utilizing the following principles: ease of manipulation by untrained personnel, adaptation to various types of clinical photography, use of commercially available equipment, and economy of materials and construction.

The equipment consists primarily of mounting brackets of appropriate length for standard or long focus lenses, to which are attached the following.*

1. Still camera, 35 mm.
2. Electronic flash lamp of the "around the lens" type with reflector and alternative off-axis mounting for special use, such as external eye photography, and when the camera view finder is required.

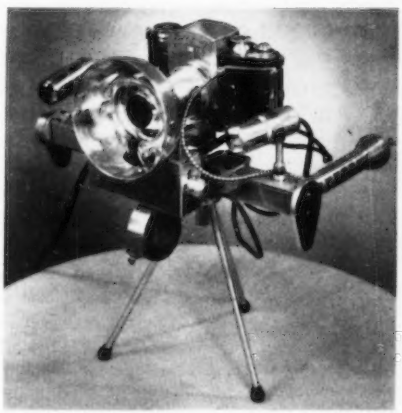

FIG. 1—Electronic flash attachment with the Exacta V camera.

3. Surgical photographic attachment consisting of two high-intensity lamps with calibrated control; the spots of light, when superimposed, indicate that the camera is properly

From St. Luke's Hospital, Chicago.
Presented as a *New Instrument* at the Fifty-Sixth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 14-19, 1951, Chicago, Ill.
*Produced by the Pho-Tak Corporation, Chicago, Ill.

centered, and that it is at the proper distance for sharp focus; special handles with thumb-activated cable release are attached.

4. Pull-out type tape measure for measuring distances.
5. Framing devices which outline the field and indicate the proper distance.
6. Tripod or pistol grip for holding.

The flash lamp is activated by the commercially available Thrift Light power pack of 100 watt-second capacity. Although this bears the seal of the Underwriters Laboratory, the equipment is not approved for use in an explosive atmosphere, so it is not safe in the presence of flammable anesthetic agents. A delay may be included for use with cameras synchronized for conventional flash bulb operation.

For surgical photography, the focusing lamps are adjusted before use so that the spots of light are superimposed at the proper distance for the particular lens. The camera is held by a nurse and pointed at the field. The surgeon directs the position of the camera so that the two spots of light are superimposed on each other at the center of the field and directs the exposure to be made. The film is advanced, and the camera is ready for the next photograph.

Kodachrome, daylight-type film gives good color, and the exposure varies from F8 to F22 depending upon the size of the field and the lens. Black and white film may be used, although under some conditions a neuter filter is required to reduce the amount of light.

The camera may be used for general clinical photography of patients, parts of the body, specimens, etc. The axial type of illumination is adequate for photographs of the mouth and oral pharynx and of the deep portions of surgical wounds. It is not designed for endoscopic photography. A 75 mm. lens has been found most useful for surgical procedures because it gives a convenient size of field at a distance which does not jeopardize the sterility of the operative field. Except for the latter, the usual 50 mm. lens is satisfactory. Extension tubes or portrait attachments may be used.

PERIPHERAL IRIDECTOMY IN NARROW ANGLE GLAUCOMA

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CHICAGO, ILL.
and
HAROLD G. SCHEIE, M.D.
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IT IS the purpose of this paper to offer an explanation for the mechanism of peripheral iridectomy in narrow angle glaucoma, and to emphasize the value of the operation. We believe that peripheral iridectomy functions by eliminating the mechanism responsible for so-called physiologic iris bombé with resultant deepening of the angle of the anterior chamber. The various interpretations of its mode of action will be reviewed.

Opinions differ as to the value of peripheral iridectomy; this, it is believed, has largely resulted from a failure to limit its use only to suitable cases of narrow angle glaucoma. This failure is largely due to a misunderstanding of the terms wide angle glaucoma and narrow angle glaucoma. Open angle glaucoma might be a more satisfactory term to describe wide angle glaucoma (chronic simple glaucoma) since it is well known that this disease occurs in eyes with angle depth varying from wide to very narrow. The term wide angle glaucoma only implies free access of the aqueous to the trabeculum-canal of Schlemm mechanism. This type of disease is often distinguishable from true narrow angle glaucoma only after long and arduous study.

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Presented at the Fifty-Sixth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 14-19, 1951, Chicago, Ill.

As used in this paper, narrow angle glaucoma will be defined as an elevation of the intraocular pressure resulting from a blockage of the trabeculum-canal of Schlemm mechanism, by contact with the iris root, due to one or more secondary factors superimposed on a preexisting shallow angle. This lengthy definition excludes from the scope of this paper those instances of wide angle glaucoma in which the angle is markedly narrow anatomically, but in which the aqueous has access to the trabeculum-canal of Schlemm mechanism. Such cases do confuse, but need not serve as an insurmountable obstacle to the concept of narrow angle glaucoma as defined by Rosengren,[11] Barkan,[2] Sugar,[15] and Kronfeld and McGarry.[10]

Iridectomy was first successfully used as an antiglaucomatous operation by Albrecht von Graefe[6-8] in 1856. During the ensuing years it has become the operation of choice for acute narrow angle glaucoma. Peripheral iridectomy for narrow angle glaucoma was introduced, to the best of our knowledge, by Bänziger[1] in 1922. In our experience both are effective by the same mechanism and should be employed only under the following circumstances: (1) during the first 36 hours of the first attack or (2) during the interval phase between attacks if the tension remains normalized with or without miotics. When these rigid indications are scrupulously observed it is the writers' opinion that peripheral iridectomy is the most satisfactory of the antiglaucomatous operations,

and in the authors' experience, results in the successful control of intraocular pressure in a very high percentage of instances.

The many explanations as to the mechanism by which iridectomy functions reveals no parallelism to the general agreement of its efficacy. Several theories have been suggested including the following: (1) von Graefe[6-8] suggested that removal of a portion of the iris diminished the amount of aqueous secreted; (2) de Wecker[4] and Priestley Smith[13] believed that iridectomy functioned through the formation of a filtering cicatrix; (3) Troncoso[16] thought that iridectomy provided a communication between the anterior and posterior chambers but that it was not necessary to re-establish drainage through the normal outflow channels; (4) François[5] supposed that the tension was modified through a nervous mechanism acting upon the secretion of aqueous; (5) Weber[19] and Sugar[14] believed that part of the closed angle was opened by iridectomy and the tension reduced by drainage through the normal channels. Sugar stated that when the operation was done after dense adhesions had formed, it was unsuccessful except when filtering cicatrices developed and (6) Bänziger[1] theorized that iridectomy might function by providing a safety valve which prevented an increase in pressure in the posterior chamber. He believed that acute attacks of glaucoma resulted in eyes with a shallow anterior chamber when a sudden increase in pressure behind the iris diaphragm forced the root of the iris into contact with the cornea. To prove this, he performed a small nonbasal peripheral iridectomy in an eye with intermittent attacks of narrow angle glaucoma and found that the attacks ceased.

The writers' opinion of the mechanism of acute glaucoma and of peripheral iridectomy is not in agreement with that of Bänziger, although our findings corroborate his observations regarding the efficacy of the operation in this type of disease. In an attempt to clarify the situation the writers have pursued the course chartered by Bänziger. For six years we have been using the peripheral iridectomy in nearly all cases which presented the indications described in the early part of this paper. This iridectomy is performed under a good sized conjunctival flap, using a 6-0 chromic catgut suture to close the scleral wound. An extremely small peripheral iridectomy is performed with no attempt to make it basal. Often the iridectomy is performed laterally or below if there is any question as to the accuracy of the diagnosis.

All eyes were examined gonioscopically preoperatively and postoperatively. At times it was impossible to observe the angle during the acute phase of an attack, even with the use of glycerin, but an attempt was always made. Whenever the angle could be visualized during an acute attack, it was closed, at least gonioscopically. In the intervals between attacks, one could correlate the gonioscopic picture with the intraocular pressure. In nearly all eyes where the pressure was normal, with or without miotics, the angle appeared open in at least one quarter of its circumference and in all instances where the intraocular pressure was high the angle appeared closed. Following successful peripheral iridectomy, the angle of the anterior chamber was deepened gonioscopically, usually along its entire circumference and could readily be adjudged open. When one could see the second portion of the trabeculum in an area corresponding to one third of the circumference, a successful result was practically assured and subsequent mydriasis tests were negative. When the second portion of the trabeculum was not visible the operation did not result

in lowering of the intraocular pressure unless evidence of external filtration was visible. These findings occurred irrespective of the size or location of the iridectomy.

In the authors' opinion these findings are extremely illuminating. The negative results obtained by excising a piece of iris[1] when the angle remains closed tend to contradict the theory that removal of iris tissue is responsible for the fall in tension, and the absence of a filtering cicatrix in the vast majority of our cases refutes the filtration theory. The use of an extremely small peripheral iridectomy makes unlikely the possibility that the iridectomy itself opens the angle over a sufficient area to effectively lower tension. Also the gonioscopic observation that the angle deepens throughout rather than in relation to the iridectomy opposes the theory that pulling on the iris opens the angle by rupturing of the peripheral anterior synechias.

The more recent belief that a nervous mechanism results in the lowering of pressure is refuted by the following series of events in one patient. This patient showed the classical picture of narrow angle glaucoma since, under miotics, the second portion of the trabeculum was visible over one half of its circumference. During these conditions the tension remained below 20 mm. Hg Schiotz. Under periods of emotional stress, or when the miotics were stopped, it could be seen gonioscopically that the angle closed and the tension rose to hard. Therefore a small peripheral iridectomy was performed at 2 o'clock by the method previously described. On the first postoperative day, the tension was found to be 80 mm. Hg Schiotz and the site of the iridectomy could not be demonstrated by direct observation or with the slit lamp. Gonioscopy revealed that the iridectomy had become incarcerated in the wound and was no longer patent. On miotics the tension again fell to below 20 mm. Hg Schiotz with a persistence of the same gonioscopic picture. Another peripheral iridectomy was performed and this time it remained patent. The tension has remained controlled below 20 mm. Hg Schiotz ever since and it is impossible to precipitate an acute attack by the use of mydriatics. Thus, one can say that the excision of a piece of iris in itself will not control the pressure through axon reflexes. Nor will a paracentesis control the pressure by breaking up a vicious cycle. It is necessary to establish a communication between the anterior and posterior chambers to control the intraocular pressure.

SUMMARY

The writers observed independently that the angle of the anterior chamber deepened throughout its circumference following peripheral iridectomy and each postulated that it was necessary to postulate a resistance to the forward flow of aqueous between the iris and the anterior surface of the lens which produced a physiologic iris bombé with narrowing of the angle. The impediment to the flow of aqueous which produces this anterior bowing of the periphery of the iris could be a close contact between the lens and iris. An acute attack of glaucoma in an eye with an anatomically narrow angle could therefore result whenever the iris was pushed forward sufficiently to come into contact with the trabeculum. The amount of bowing of the iris wall might vary depending upon the degree of obstruction to the aqueous flow from the posterior to the anterior chamber, and conceivably upon the rate of formation of aqueous. Peripheral iridectomy would be effective in deepening the anterior chamber, therefore, by eliminating the resistance to the forward flow of aqueous and by lowering the pressure exerted on the posterior surface of the "physiologic iris bombé."

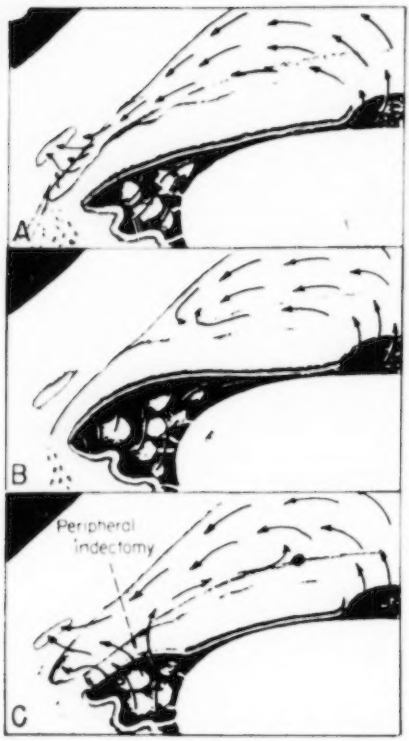

FIG. 1.—A. Aqueous passing through pupil to reach trabeculum in an anatomically narrow angle (tension normal). B. During acute attack aqueous passes through pupil but can not reach trabeculum due to iris root block (tension hard). C. Following peripheral iridectomy there is over-all deepening of anterior chamber (tension normal).

This concept of physiologic iris bombé receives additional support from the earlier observations of Ulbrich[17] who reported in some detail the findings in an eye with a small hole in the iris covered by a very thin loose membrane. This membrane bulged into the anterior chamber under normal conditions indicating that under ordinary circumstances the pressure in the posterior chamber is greater than that in the anterior chamber. Heine[9] similarly reported a case with a congenital umbrella iris in which posterior synechias were absent, the condition being due to aplasia irides. He concluded that there was a slightly higher pressure in the posterior chamber which caused a bulging forward of the umbrella iris.

In addition to the pressure inequality, there is evidence that the impediment to the flow of the aqueous can be enhanced by the use of miotics. Urbanek,[18] in a child with high grade congenital aplasia of the crypt layer (Kryptenblatt) of the iris, demonstrated that with maximal myosis and in the absence of accommodation the pressure in the posterior chamber is increased. The membrane bulged in a hernia-like manner from the limiting ring into the anterior chamber. Thus he concluded the outflow of aqueous humor through the pupil must be interrupted at least until a certain pressure is reached in the posterior chamber. The more recent work of Scheie[12] and Barkan[3] with air-induced glaucoma also substantiates the concept that a close contact between the iris and lens can cause iris bombé. Thus it is the opinion of the writers that peripheral iridectomy functions by deepening the anterior chamber due to a short circuiting of the aqueous past the resistance of the iris, which maintained a higher pressure in the posterior chamber than in the anterior chamber. This mechanism must also be included among the mechanisms considered to produce or at least contribute to anatomically shallow angles (fig. 1).

CONCLUSIONS

The writers reaffirm the effectiveness of peripheral iridectomy in narrow angle glaucoma of the acute type. The operation successfully controls ocular tension without miotics in nearly all instances if the eyes are properly selected. Successfully operated eyes then respond in a totally normal fashion and are negative to all of the provocative tests for narrow angle glaucoma, including mydriasis.

The writers believe that peripheral iridectomy functions by correcting the obstructive mechanism responsible for physiologic iris bombé. By creating a hole in the iris that structure is permitted to drop backward and protect the eye against future acute attacks of glaucoma resulting from contact between the iris and trabeculum.

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DISCUSSION

DR. PAUL A. CHANDLER, Boston, Mass.: This paper by Dr. Haas and Dr. Scheie is an important contribution to the treatment of narrow angle glaucoma. It emphasizes the value of careful observation, and once again makes it clear that the clinical field is not yet entirely gleaned. The concept that there is a greater degree of obstruction to flow from posterior to anterior chamber in eyes with shallow anterior chambers than in those with normal chambers was advanced by Curran (Arch. Ophth. 49:131-155, 1920) and was the basis for his operation of peripheral iridotomy. Curran noted the deepening of the periphery of the anterior chamber after peripheral iridotomy and felt that it could only be due to the opening up of free communication between posterior and anterior chambers. Curran's operation fell into disrepute, since without the gonioscope he could not recognize that the angle was permanently closed in the more advanced stage of narrow angle glaucoma and his operation could not be effective in these cases.

In 1922 Bänziger stated his belief that a relative pupillary block existed in eyes with shallow anterior chambers and advised peripheral iridectomy for acute glaucoma. With the advent of the gonioscope the concept that acute narrow angle glaucoma was due to mechanical blocking of the angle was firmly established. Barkan[2] presented the first classification of primary glaucoma based on gonioscopic observations and advised multiple peripheral iridectomies in narrow angle (iris block) glaucoma.

The authors of this paper have presented the first clear-cut statement of the indications for peripheral iridectomy. My own experience fully corroborates their views. The operation will obviously be ineffective if the glaucoma is advanced and a large part of the angle is permanently closed by peripheral anterior synechias. I can also testify to the effectiveness of peripheral iridectomy or iridotomy in the more chronic form of narrow angle (iris block) glaucoma, if done early in the disease.

At what stage in chronic narrow angle (iris block) glaucoma will this simple operation be effective? A great deal more clinical experience is necessary to fully answer this question. If the tension can be reduced to normal with miotics, the chances are favorable for the operation to be effective, even though slight cupping of the disc and limited field changes are present. One of the great advantages of peripheral iridectomy or iridotomy over full classical basal iridectomy is in the visual results. After classical basal iridectomy, no eye is ever functionally the same as before. Often there is irregular astigmatism and though vision with lenses may be recorded as 20/20, the vision is never so clear as before. After peripheral iridectomy or iridotomy the eye is functionally fully normal. Furthermore the complications of full basal iridectomy are much greater than those of peripheral iridectomy. I have had a limited exprience with the Curran peripheral iridotomy. I have found it technically difficult, but when successfully performed there is minimal trauma to the eye and the results are in every way comparable to peripheral iridectomy. The authors advocate suture of the scleral wound in peripheral iridectomy and this is a point that deserves emphasis. If after the operation, due to a leaking wound, the anterior chamber remains flat for twenty-four hours or more, the anticipated results of the operation may be completely nullified due to the formation of peripheral anterior synechias.

No one who has observed the angle of the anterior chamber gonioscopically in these cases before and after peripheral iridectomy or iridotomy can fail to be impressed by the results of a tiny opening in the periphery of the iris. Indeed one does not need a gonioscope to note the difference in contour of the iris from a smooth forward bowing before operation to a flat contour afterward. I believe with the authors that there is no other tenable explanation for this change in contour of the iris and the complete clinical cure of this type of glaucoma by making a small opening in the periphery of the iris than the relief of a relative pupillary block or physiologic iris bombé by eliminating obstruction to flow of aqueous from posterior to anterior chamber. The relative safety of peripheral iridectomy should influence our treatment of the fellow eye in cases of acute narrow angle glaucoma. I now advise prophylactic peripheral iridotomy or iridectomy for the fellow eyes of all young and middle aged patients with acute glaucoma in one eye.

Peripheral iridectomy or iridotomy is not to be regarded as a temporary arrest of glaucoma as is the case with filtering operations in wide angle glaucoma, but as a permanent and complete cure of a certain type of glaucoma, providing there are no operative or postoperative complications and if done according to the indications the authors specify.

Let us hope that such a cure for other types of glaucoma will be discovered in the not too distant future.

DR. S. RODMAN IRVINE, Beverly Hills, Calif.: The authors' observations on certain cases of acute narrow angle glaucoma lead them to believe that the immediate etiology in these cases is a pupillary block mechanism. This produces a functional iris bombé, the iris being displaced forward and occluding the iridocorneal angle.

Now since a small peripheral iridectomy or iridotomy deepens the chamber and relieves the glaucoma the authors' concept of the physiologic pathology seems to be substantiated.

The same type of problem occurs when herniated vitreous blocks the pupil, as has been described by Chandler and Reese. Similarly Scheie has called our attention to pupillary block by air injected into the anterior chamber after the cyclodialysis operation.

I would like to point out that the concept of pupillary block in certain types of glaucoma has been taught by Chandler for a

number of years and now it seems that Drs. Haas and Scheie have given additional proof for this concept.

I believe the iris must bind the lens in such cases preventing escape of aqueous between the iris and the lens.

I cannot understand why constriction of the pupil does not result in further pupillary block, as it does when herniated vitreous occludes the pupillary space. Perhaps the lens is flatter at the anterior pole so that contraction of the pupil lessens the binding effect between iris and lens and at the same time pulls the iris away from the angle.

Now, if the anterior pole of the lens is convex as the result of swelling, contraction of the pupil would increase the pupillary block and the tension would increase with increased miosis, and one might be misled in the diagnosis of acute narrow angle glaucoma susceptible to cure with iridotomy. Nevertheless, iridotomy would work by deepening the anterior chamber just as it does in narrow angle glaucoma that responds to miotics. Of course, if the lens is so large as to block the anterior chamber regardless of miosis or iridectomy, one is dealing with malignant glaucoma and the lens must be removed.

In the type of case under discussion I would suggest the possibility of transfixation of the iris with a cataract knife to establish communication between the posterior and anterior chambers rather than turning down a conjunctival flap and making an incision as suggested by the authors. In case of failure of the peripheral iridotomy resulting from transfixation of the iris in this way one would still have normal conjunctiva preserved for a subsequent filtering operation. Also the chance of anterior chamber hemorrhage would be lessened.

Dr. Scheie and Dr. Haas are to be congratulated for their work and ideas which show so much ingenuity and imagination, not only for the present paper but for work leading up to it.

DR. SCHEIE: Dr. Haas and I are extremely grateful for the kind treatment accorded us by Dr. Irvine and Dr. Chandler.

The reason for our presentation is first to emphasize the importance of peripheral iridectomy. I want to be perfectly frank in stating that a few years ago I first did the operation after being urged to do so by Dr. Kronfeld and Dr. Haas, while visiting their glaucoma clinic.

After observing a few cases postoperatively with a gonioscope I noticed that the anterior chamber had deepened as a result of the procedure, and upon comparing notes with Dr. Haas I found that he had made an identical observation. As a result, we offered this joint presentation.

I would like to emphasize the importance of observing the indications pointed out in the paper and also mentioned by Dr. Chandler.

I agree entirely that suture of the wound is very logical, not that I have done it as consistently as Dr. Haas has, but I am going to use it more consistently in the future.

I would also like to elaborate just a bit upon a remark made by Dr. Irvine who suggested that the primary cause of acute narrow angle glaucoma might be the physiologic iris bombé. I am sure that he meant to say that this was the precipitating or secondary cause, the primary or underlying cause being the anatomically narrow angle that is probably an individual anatomical characteristic.

MICROWAVE DIATHERMY IN OPHTHALMOLOGY: THE VARIOUS DIATHERMY CURRENTS USED IN OPHTHALMOLOGY

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THE PURPOSE of this paper is to discuss the relative position in the electromagnetic spectrum of the various diathermy currents used in ophthalmology. Diathermy currents are termed so because they induce heat in certain substances. We are especially interested in them because of their application to induce heat in body tissues. For purposes of this discussion, the induction of general systemic fever will be omitted, and only currents used locally for elevating the temperature of ocular and local orbital tissue will be considered.

For our purposes these diathermy currents may be classified as follows: (A) medical diathermy—useful for the production of passive moderate rises in tissue temperature (rise of 4.5° to 7.5° F., or 2.5° to 4° C.) and (B) surgical diathermy—useful for various degrees of destruction or for the cutting of tissues. (It is also used for the secondary production of inflammatory response, such as adhesive choroiditis in surgery for retinal detachment.)

Medical diathermy may be induced by the application of the so-called long waves, short waves, or microwaves of the radio frequency spectrum.

It is evident that the multitude of frequencies in use by communications of various types, by the broadcasting and by television industries, and by medical diathermy, required the assignment of definite channels for various uses by international and federal regulating bodies. This was necessary to avoid interference.

The Federal Communications Commission (F.C.C.) has assigned definite waveband frequency channels for medical short-wave diathermy. These are 13.66, 27.32, and 40.98 megacycles. It has also assigned several channels for microwave diathermy, namely 915, 2,450, 5,850, 10,600, and 18,000 megacycles. Manufacturers are now licensed to produce apparatus which adheres to these frequencies without significant variations.

The long waves that could be used in medical diathermy vary in wave length between 3,000 meters and 30 meters. The actual available apparatus affords a wave length of 600 and 300 meters. This corresponds to a frequency* of 500 and 1,000 kilocycles. They are sometimes known as Hertzian waves, or high frequency current.

Long-wave diathermy is produced by the spark-gap type of machine. Its frequency is sufficiently high to avoid tetanizing stimulation of muscles. Large metallic, flat electrodes must be kept in good contact with the skin to avoid local arcs or burns and to produce a proper tissue temperature rise. Long-wave diathermy was introduced about 1922.

About 1930, a short-wave band of the radio frequency spectrum was adopted for medical diathermy by some workers. It utilized the so-called short wave at approximately 30 meters wave length, having a frequency of 10,000 kilocycles,

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* 1 kilocycle frequency = 1,000 cycles per second. 1 megacycle frequency = 1,000,000 cycles per second.

or the so-called ultrashort waves having a wave length of approximately 6 meters, or a frequency of 50 megacycles.*

Short-wave and ultrahigh frequency are produced by an oscilating circuit containing one or more vacuum tubes (triode).

The short-wave medical diathermy machines on the market use 7.3, 11, or 22 meter wave lengths at an approximate frequency of 41 megacycles, 27.3 megacycles, and 13.66 megacycles. Short-wave diathermy does not require electrode skin contact. It induces a tissue heat effect with the aid of cuffs or air-spaced electrodes which produce an "electric field." On the other hand, it can be applied with the aid of drums or coils to induce an "electromagnetic field." Both types of field result in tissue heating. The short-wave form produced by the vacuum tube circuit is undamped, that is, the form of each wave is uniform and continuous. On the other hand, the waveform produced by the spark-gap circuit is damped, that is, the waves come in groups of spurts of progressive reduction in amplitude and energy (table I).

Shortly after World War II, it was found possible to utilize radar waves for inducing rises in tissue temperature. These waves belong to the microwave section of the electromagnetic spectrum and include wave lengths between 1 millimeter and 1 meter. They correspond to frequencies between 300,000 megacycles and 300 megacycles. At the present time, the available apparatus for microwave diathermy produces oscillations of 12.2 cm. wave lengths, having a frequency of 2,450 megacycles. Microwaves have properties of linear propagation and can be beamed, reflected, refracted, and focused. They have advantages over infrared wave lengths or frequencies because of their deeper penetration without excessive surface heating. The production of these extremely short waves was made possible by the invention of the multicavity magnetron in Great Britain about 1940. (The mass production of this extremely high-frequency oscillator, required in such great quantities during World War II, is a tribute to American engineering ingenuity.)

TABLE I

WAVE LENGTH AND FREQUENCY OF DIATHERMY CURRENTS

TYPES	WAVE LENGTH RANGE (Available apparatus)*	FREQUENCY RANGE
Long-wave (Spark-gap)	3,000 - 30 m. (600 - 300 m.)	100 - 10,000 kc. (500 - 1,000 kc.)
Short-wave (Vacuum tube)	30 - 3 m. (30 - 6 m.)	10 - 100 mc. (10 - 50 mc.)
Microwave (magnetron)	100 cm.-1 mm. (12 cm.)	300 - 300,000 mc (2,500 mc.)

* Comments in parenthesis have reference to current types of apparatus, and wave lengths and frequencies produced by them.

The Microtherm made by the Raytheon Company is a microwave generator employing a multicavity magnetron with an input of approximately 100 watts of energy. The generated microwaves of 2,450 mc. frequency are channeled by a flexible coaxial cable connected to any one of three chosen directors. These directors are used for focusing beams of microwaves to induce diathermy in tissue. They are usually kept at a distance of 2 to 3 inches from the skin. Director B is used for treating large areas. It produces a tubular concentration pattern of microwaves. Director A also produces a tubular concentration pattern of microwaves. Director C produces an oval conical concentration pattern.

Diathermy used by the above modality induces a local rise in temperature in the orbital or ocular tissues because they offer a resistance to the passage of the electromagnetic waves. It is likely that chemical processes are somewhat accelerated by the temperature rise. Microwaves have been shown to cause an increase in blood flow, which can dissipate relatively excessive heating. On the other hand, the vitreous and lens possessing no blood circulation may become overheated.

Excessive concentration of microwave energy produced by (1) large wattage input, (2) improperly designed directors, (3) improper pointing of such directors, (4) too close approximation of microwave directors to skin surface, and (5) prolonged application of this modality, can produce dangerous overheating of tissues. Director C is preferred in ophthalmology. The smaller hemispherical director can also be used in treating scleritis, keratitis, iridocyclitis and other nonexudative inflammations. They should not be used closer than two inches.

Because the Microtherm has an approximate output of 100 watts, the full output is unsafe for ophthalmic use. It can, however, be regulated quite readily by its controlling dial. Twenty per cent of the output applied with a C Director at a distance of two or three inches from the skin surface of the closed lid or from the lateral orbital surface for a period of 20 minutes, appears for the present to be a safe procedure, avoiding overheating of delicate ocular structures and undesirable sequelae.

Microwave diathermy is now being applied in iritis, cyclitis, scleritis, keratitis, inflammatory lesions of the lids and other inflammatory conditions of the orbit. It may have promise in the absorption of exudates and hemorrhages and in the production of vasodilatation which is advisable in some degenerative retinopathies.

Microwaves and other diathermy are generally contraindicated in (1) cases with bleeding tendencies, (2) over metallic implants, (3) in the presence of local malignancy, (4) in the presence of headache of cerebral origin, (5) on anesthetized areas, (6) over moist dressings, and (7) over adhesive tape.

SURGICAL DIATHERMY

Surgical diathermy utilizes long or short waves. The usual wave length of surgical currents varies between 27 meters and 200 meters.

Essentially three types of wave form are used: (1) the damped wave form, or coagulating current, for coagulation, fulguration, desiccation of tissue, and hemostasis, (2) the undamped wave form or cutting current, for biopsy, surgical incision, epilation, and (3) the blended current, a combination of the above two when cutting current requires coagulating qualities.

The damped wave is produced by the spark-gap type of machine and consists of rapid repeated bursts of wave groups which decrease in amplitude as the burst of energy discharges.

The undamped waves of the surgical diathermy machine are produced as in medical diathermy by an oscillating circuit containing one or more vacuum tube circuits.

The applicator or electrode in surgical diathermy is relatively small and concentrates heat at the zone of contact with tissue.

Surgical diathermy may be applied with a single electrode. This is known as the unipolar method. It is used for fulguration or sparking tissue destruction, or for desiccation with the sharp electrode buried in tissue. It is preferably applied interruptedly to avoid overheating.

The above technics are also used together with a large indifferent electrode applied to the back, arm, leg, or shoulder.

Coagulation is produced by the bipolar method using damped waves. A ball electrode is applied for surface coagulation with the indifferent electrode applied to the shoulders. This is the method for scleral surface coagulation in flat retinal detachments. On the other hand, bipolar coagulation between two small, sharp and closely spaced electrodes is used for destruction of tissue and localized current effect.

The blended current is valuable for making scleral micropunctures in the diathermy operation for retinal detachment, either by the Safar-Walker, or the Weve needle technic, for cyclodiathermy in advanced glaucoma and for procedures in which only moderate destructive effect is desired. Some surgeons, however, use reduced, damped, "coagulating" currents for this purpose.

SUMMARY

1. Long-wave diathermy waves have a frequency of 500 to 1,000 kilocycles and a wave length of 600 to 300 meters. They are generated by spark-gap apparatus.

2. Short-wave diathermy waves have a frequency of 10 to 50 megacycles and a wave length of 30 to 6 meters. They are generated by vacuum tube oscillating circuits.

3. Available microwave diathermy has a frequency of 2,450 megacycles and a wave length of 12.2 centimeters. They are generated by the multicavity magnetron circuit. Care must be taken in the use of microwave diathermy to keep the wattage at 20 per cent of the machine output, to keep directors at 2 to 3 inch distance from skin, and to keep the time of treatment at a maximum of 20 minutes.

4. Surgical diathermy uses the damped wave for hemostasis, coagulation, desiccation, or fulguration, and the undamped wave, as a cutting current.

5. The blended current combines the cutting properties of the undamped wave with the coagulating properties of the damped current.

MICROWAVE DIATHERMY IN OPHTHALMOLOGY: CLINICAL EVALUATION

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I HAVE no doubt that most ophthalmologists in this audience have had the same experience that I have had in the management of many disease conditions in the posterior ocular segment. My own experience has been, in general, extremely discouraging. For that reason I have been inclined, as I suppose you have, to welcome any new method that seemed to promise better results. I am afraid that I have also been inclined, at least until experience taught me otherwise, to grasp at any improvement observed, without too much critical evaluation. We all seem to be avoiding that particular error in microwave diathermy. Careful animal experimentation has preceded clinical testing. The physiologic implications of the experimental results have been carefully studied. Finally, there has been an admirable degree of restraint in the attribution of clinical successes to this method. I am sure, therefore, that I need not emphasize to this audience that the experiences which I am about to relate constitute merely a clinical report, and a preliminary one at that, and that nothing more than that should be read into them.

PREVIOUS METHODS

My interest in treating diseases of the posterior ocular segment by elevation of the regional temperature goes back to the late 1930's, when I attended an instructional conference at one of the Academy meetings and heard the late Dr. Clifford Walker recommend conventional diathermy for the management of early secondary senile macular degeneration (which I shall, for convenience, refer to henceforth merely as senile macular degeneration). His recommendation was made on the ground that dilatation of the choroidal blood vessels might conceivably be helpful. Although we followed Walker's technic in every detail, our personal results with this method were no better than results with methods which we had already discarded as ineffective.

Our next approach to the problem was prompted by the observation that many patients with early senile macular degeneration also presented mild to moderate secondary anemia. The observation was made often enough to warrant the assumption that a nutritional deficiency in the macular region, which is the presumptive cause of this disease, might be explained by anoxia, which might be explained, in its turn, by a red blood cell deficit. Acting on this assumption, we employed all the standard therapeutic measures for anemia, including transfusions of whole blood and of red blood cells, but again the results were disappointing. Macular degeneration was not influenced even when the red blood cell values were elevated above normal, and even when the patients were seen early.

We then turned to the use of vasodilators. The nitrates and nicotinic acid proved too fleeting in their action to be useful. Various mercurial drugs were equally ineffective. Priscoline proved unsafe; its generalized vasodilator ac-

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tion caused visceral bleeding in several cases. Depropanex was the most satisfactory of all the therapeutic agents employed, and, as I shall point out shortly, we have continued to use it as an adjunct measure, no matter what other methods of therapy we have employed.

MICROWAVE THERAPY

As soon as microwave diathermy became available, late in 1947, we began to study it, first experimentally, and then by cautious clinical use. On the whole, our results have been promising, and, what seems quite as important in a method still undergoing clinical testing, we are quite certain that we have not done any harm in any case in which it has been used.

The approximately 75 cases upon which this communication is based include senile macular degeneration, retrobulbar neuritis, central serous retinopathy, thrombosis of the central retinal vein and its branches, traumatic hemorrhage, and postoperative uveitis. All of these patients were treated by a standardized technic. We have excluded from consideration all of our early cases, in which we were working out dosages and methods, though it seems worthwhile to say that we are quite certain that we did no damage in any of them either. It also should be noted that all of the cases in this series are instances of a single disease condition; we have not included in this evaluation any case complicated by multiple diseases.

Technic

Unless otherwise stated, all the patients in this series were treated by a technic which we are still employing and which we have worked out by a process of evolution. The triangular applicator of the Raytheon Microtherm is used at a distance of 3 inches from the temple, with the output directed to the region of the choroid. With this technic the heat is concentrated within an area of 1 to 2 inches. Each treatment lasts for fifteen minutes and utilizes 20 per cent of the output of the machine. As long as the patient is under treatment he takes nicotinic acid and thiamine chloride by mouth, the former in 50-mg. doses four times daily and the latter in 25-mg. doses by the same scheme. He receives Depropanex (2 cc.) intramuscularly before each treatment.

The first course of microwave diathermy consists of three treatments weekly for three weeks. At the end of this period the visual acuity and central fields are checked against the pretreatment record. If there has been no improvement in vision or if, as sometimes happens, there has been a deterioration, it is usually concluded that microwave diathermy will not be effective in the special case and it is discontinued. If the visual acuity has been maintained at the original level or has improved, the same regimen of therapy is repeated for another three weeks, after which another re-evaluation is carried out. Our experience has been that if treatment is to be successful, maximal benefits will usually be observed by the end of the six-week period. However, in some instances additional improvement in visual acuity may be observed to take place over a longer period of time.

We have found that patients vary in their response to treatment. The length of remissions differs from case to case, and so does the amount of treatment required to maintain whatever benefit may have been received. When maximal benefit has been achieved, therefore, the procedure is adapted to the reaction of the individual patient. Until this can be determined, we reduce the number of treatments by a fixed routine. They are reduced from three to two a week for six weeks, and then to one a week for the same period of time. Visual acuity and central fields are checked at the end

of each six-week period. The patient is then dismissed, with instructions to return for examination at the end of six weeks unless he notices any decrease in visual acuity earlier; if he does, he must return at once. We have learned that some patients, in order to maintain whatever benefits they have received, must be treated at weekly intervals. Others require even more frequent treatments, while still others can go for longer periods. The essential thing is to learn the pattern of the individual response to therapy and to treat the patient accordingly.

One thing we have learned from our experience with diseases of the posterior ocular segment is that we must alter our criteria of success. In senile macular degeneration restoration of vision does not seem to be an attainable objective. We have found that we cannot help any patient who comes to us with vision of less than 20/40. This is the poorest vision with which ordinary print can be read without the aid of a magnifying glass or telescopic lens. We therefore regard the maintenance of vision at a useful level, that is, 20/40 or better, as the criterion of successful treatment in senile macular degeneration and other diseases which we have treated with microwave therapy. To express it differently, our objective is the prevention of further regression, and we feel that we have accomplished a good result when we have brought the disease to a standstill.

CLINICAL RESULTS

Senile Macular Degeneration

Our first, and widest, experience with microwave diathermy has been in the treatment of senile macular degeneration. By the criterion which I have just described, I think it fair to say that our results have been encouraging. They are the more encouraging when one realizes that almost without exception the patients who experienced no improvement came to us with vision below 20/40, that is, with vision that had already sustained an irreversible decrease.

If I have arrived at any important conclusion from this clinical experience, it is that as ophthalmologists it is our duty to try to do something for patients with secondary senile macular degeneration, all of whom are elderly, before their vision reaches the point at which we can do nothing for them. We ought to remind ourselves, and we ought to teach our students, that it is simply not enough to tell a patient who complains of a drop in visual acuity that cannot be improved by refraction that he has an incipient cataract. We all know that presenile changes in the lens, such as cuneiform and coronary opacities, lamellar separations, and water clefts in the suture lines do not produce marked decrease in visual acuity. We all know that patients with so-called incipient or presenile cataracts can be kept with 20/20 vision for years merely by changing their glasses as the refractive index of the media changes. In short, a patient whose fundus can be seen clearly with an ophthalmoscope and whose vision cannot be improved to normal with refraction deserves a central field examination.

Retrobulbar Neuritis

I hope that some day, and that in the not too distant future, the neuro-ophthalmologist will present us with some perimetric method of making the differential diagnosis between early senile macular degeneration and retrobulbar neuritis. By present methods the field defect, a small central scotoma, seems the same in both. The most intensive study provides no differential help. In our own clinic we use the binocular ophthalmoscope with Hildreth ultraviolet light and filters, the Friedenwald ophthalmoscope, and the Goldmann corneal microscope with Rhuby lens. It may be that

electroretinography and flicker-fusion fields, which we have not yet tested, will provide the diagnostic aid required.

Since this diagnostic confusion exists, it was natural that on some occasions when we thought we were treating retrobulbar neuritis, we found later that we had been dealing with early senile macular degeneration. Some patients with presumptive retrobulbar neuritis, whose vision had been greatly benefited by microwave diathermy, failed to return for examination at the intervals set. When they did return, months later, they presented frank degeneration of the macula with pigment changes and irreversible loss of vision. These cases prove the difficulty of making the differential diagnosis between the two conditions.

In spite of that confusion, our results with retrobulbar neuritis treated by microwave diathermy have been encouraging on the whole when they are analyzed by the criterion of maintenance of visual acuity at a useful level.

Central Serous Retinopathy

Although central serous retinopathy is a disease which can be picked up by objective examination, all of us know that no effective treatment for it has yet been devised. It seemed logical to treat it by microwave diathermy, on the ground that the pathologic process is a hydrops of the macular area, resulting from local failure of circulation. Our results in the 8 cases in which we have used this method have been extremely encouraging. The condition tends to recur, but patients tend to be most cooperative about reporting for treatment, because they know that after one or two weeks of microwave diathermy any loss of vision within the compensable range is likely to be corrected.

Thrombotic and Hemorrhagic Conditions

We have used microwave diathermy in a small number of cases of branch thrombosis of the retinal vein, with what can fairly be described as outstanding success. The absorption of hemorrhage, the recanalization of the vessels, and the return of function to the affected eye have been nothing less than spectacular. Unfortunately, in thromboses of the central retinal vein, in which we had expected our results to be equally good, they were just as spectacularly bad. For this we have no explanation.

The good results observed in branch thromboses of the retinal vein were, however, repeated in a few cases of traumatic vitreous hemorrhage. I am quite certain that by using this method we have saved the vision of more than one eye in which the outlook was initially poor. We began to use microwave therapy in traumatic vitreous hemorrhage with full awareness of the risk of causing more bleeding. Our feeling, however, was that this is a desperate condition, for which a desperate remedy is warranted, and the risk proved worthwhile. If the method is to be successful, it must be used early. It is not effective after organized clot formation has occurred.

Postoperative Uveitis

In the 7 cases in which we have used microwave diathermy for postoperative uveitis we have had very good results. Almost always there has been prompt relief of ciliary pain and a sense of generalized well-being, while redness and inflammation have disappeared even more rapidly than in the cases treated with topical cortisone alone. Our technic in this disease is to use 30 per cent of the output of the machine and reduce the length of the treatments, which are given daily, to ten minutes. Applica-

tions are continued until the eye is quiet, which is usually five to ten days after treatment is started.

COMMENT

The obvious advantage of microwave therapy is that with it it is possible to heat the deeper tissues without undue heating of the more superficial tissues. With the modern equipment now available it is possible to localize the heat to the special area to be treated, and to control and measure the dosage. The technic of application is simple and the patient is comfortable and free from apprehension while he is undergoing treatment.

Animal experiments and clinical results both indicate that the temperature of the vitreous can be raised to a therapeutic degree (103° F.) when microwave diathermy is used correctly. We had planned to continue our own experimental studies on cadaveric and enucleated eyes but abandoned the idea when we reflected that the loss of normal body temperature would inevitably alter the results. No harm could result from determining the temperature elevation in an eye hopelessly destroyed and about to be enucleated, but we have not yet found a patient willing to volunteer for the experiment. The thought of the introduction of the thermocouple into the eye, which would require opening the globe, and the sight of the equipment effectively discouraged the few patients who had originally shown any interest in the matter.

With the method we use, which utilizes 20 per cent of the output for fifteen minutes at a time, we have never observed any damage to the crystalline lens, although many of our patients receive very large numbers of treatments. This is a most important consideration. As you know, most animal experiments in which serious lens damage was reported were conducted with dosages considerably heavier than those used clinically. The only case of our own in which any output larger than 30 per cent was used furnishes an interesting contrast to the experimental results.

This patient, a 36-year-old woman with optic atrophy, had vision limited to hand motion. The crystalline lens was clear when she was first treated, January 23, 1950. The schedule of treatment, which was entirely for testing purposes, was as follows:

From January 23 through July 27 she received 36 treatments utilizing 20 per cent of the output of the machine. In these and all succeeding treatments the triangular applicator was directed through the dilated pupil. Between the latter date and August 24 she had six treatments utilizing 30 per cent of the output and three utilizing 45 per cent. Between the latter date and August 30 she had three treatments utilizing 60 per cent of the output.

At this time she complained of discomfort from the heat and treatment was discontinued until September 27. Between that date and October 30 she received eleven more treatments utilizing 45 per cent of the output of the machine. At this time, because of intercurrent illness, treatment was discontinued permanently.

Careful examination at regular intervals during the course of treatment, when it was concluded, and again in July 1951, eighteen months after treatment had been begun and nine months after it had been stopped, always showed the lens to be perfectly clear. Curiously, this patient's vision, which had been limited to hand motion when she was first seen, was 20/200 when treatment was discontinued and has remained at that level. We regard this improvement as entirely unrelated to microwave diathermy. We regard it as extremely significant, however, that this patient, who could be tested clinically without fear of damage because her sight was irretrievably lost, could receive 59 treatments over a nine-month period with no harm at all to the crystalline lens, and with an incidental slight improvement in vision, although 23 treatments utilized a considerably higher output of the machine than we are accustomed to use therapeutically.

SUMMARY

Microwave diathermy seems to offer a simple and rational method of treating diseases of the posterior ocular segment if maintenance of visual acuity at

a useful level (20/40) is used as a criterion of success. It has been employed with generally encouraging results in secondary senile macular degeneration, retrobulbar neuritis, central serous retinopathy, certain thrombotic and hemorrhagic conditions, and postoperative uveitis. It is also significant that with the use of what is accepted as a therapeutic amount of the output of the diathermy machine, no lens damage has been observed in any of the cases treated by this method.

DISCUSSION

DR. R. H. DAVIES, Detroit, Mich.: Dr. Clark's paper presents a very definite and rational method of treatment pertaining to diseases of the posterior ocular segment and one which most ophthalmologists will welcome as a useful tool in the realm of ocular therapeutics. The results of the cases which he has presented offer definite encouragement in the restoration of useful vision in eyes which, in many instances, have generally progressed to complete destruction. As described by Dr. Clark this form of therapy should be included as a further adjunct in the therapeutic measures employed for these ocular conditions.

In reporting his results in this series of cases it is encouraging to note that the essayist has used a standard technic in this form of therapy, and, he is to be congratulated upon employing such great effort in his methods for evaluation pertaining to the success of the treatment with microwave diathermy. It was a difficult matter for me to properly evaluate a series of 100 cases of ocular diseases recently in which microwave diathermy was employed. These cases included retrobulbar neuritis, thrombosis of the central retinal vein and/or its branches, postoperative uveitis, acute iritis, chronic uveitis, Berlin's edema, traumatic hemorrhage, postoperative hyphema, acute congestive glaucoma, infections of the lids, herpes zoster, corneal ulcers and corneal lacerations. In most instances the diathermy was used in conjunction with the local instillation of mydriatics or miotics as indicated. On the whole the results were promising and encouraging as they were in Dr. Clark's cases. Similarly, we are certain that no harm was done in any case in which diathermy was used.

In 1940 Ruedemann and Zeiter reported a series of 60 cases treated with electromagnetic diathermy. Nineteen ocular conditions were treated including central venous thrombosis, corneal ulcers, tarsitis, herpes zoster, uveitis, malignant exophthalmos, vitreous degeneration, postoperative inflammation, and acute inflammatory glaucoma. As in Dr. Clark's series the results were encouraging.

It has been shown by careful animal experimentation that exposure of the eye to microwaves when using 60 to 70 per cent of the maximal output of the Raytheon microwave generator, with the C Director (corner director) at a distance of 2 inches from the skin and exposure for twenty minutes, the temperature of the vitreous was raised to an average of 105° F. It is felt that care must be taken to employ dosages which will not exceed this temperature elevation and a technic which will not damage ocular or adnexal tissue. Experimentally using 75 per cent output with the director 3 inches from the skin for twenty minutes, the average increase in temperature of the vitreous was 2.5° C., that of the retrobulbar tissue was 1° C. and that of the aqueous was 2.4°.

Cataracts have been produced experimentally in animals using the same apparatus at an output of 94 watts at a distance of 2 inches for a ten-minute exposure. This has been done by Daily et al, at the Mayo Foundation and has been used to study the experimental production of cataracts. It is to be noted that the Raytheon Microtherm operates at a wave length of 12.2 centimeters and has a maximum output of 125 watts. Hence, the upper limits of maximal output are used in the above instance. In Dr. Clark's series of cases, and in our series of cases, 20 per cent and 40 per cent of the output of the machine were utilized respectively. In analyzing the clinical results of these cases, no lens damage has been observed. Also to be considered in this analysis and already pointed out by the essayist, the type C Director of the microwave machine was used in his series. This allows a maximum of heat at the center of the heated area. In our series of cases type A Director, a 4-inch hemisphere type, was used. With this director the temperature at the center of the heated area of the skin is 50 per cent of that at peak levels. Thus, in the use of microwave diathermy it is of value to be acquainted with the capabilities and output of your microwave apparatus and to carefully observe the response and reaction of the individual to this form of therapy. It is important to stay within the point of tolerance of the patient and not to proceed beyond the point of comfort.

DR. GRAHAM CLARK, New York, N.Y., (by invitation). As physicians we have inherited a basic right in the field of electricity, for you will remember that it was the personal physician of Queen Elizabeth of England who coined the term "electricity." He derived that from the Greek "elektros", meaning amber, which was the substance used to produce early electrical phenomena. We neglected this birthright of ours and have contented ourselves too often to gain our knowledge of the electrical equipment which we are using from the pamphlets which come with the apparatus as it is delivered to us.

Dr. Thorpe has given us an excellent summary of what is a rather complex field, and he has introduced it in simple terms. I should like to re-emphasize one point which Dr. Thorpe has brought up in order to illustrate a need for basic knowledge of the fundamentals of electricity. There are two instruments on the market today, both of which are accompanied by brochures highly recommending them for use in treatment of retinal detachment. Examination of the machines and the circuits, if one is wont to do so, will show very little dissimilarity. The only detectible dissimilarity is the fact that the voltage-amperage ratio is reversed, the first machine producing what we call the fulgurating or desiccating current and the second machine producing what we call the coagulating current, the one that we require in the treatment of retinal detachment. The first machine is excellent for removing warts or for doing transurethral resections of the prostate but it is not for cases of retinal detachment.

Dr. Clark's paper is certainly interesting in that it gives us another insight into the important field of electricity in medicine. The field of high frequency current is practically limitless in its variations and effects and this type of microwave therapy will be investigated further as it certainly deserves.

Question may well be raised, of course, in the cases presented in Dr. Clark's paper as to whether the improvement was derived from the microwave therapy or from the vasodilating and nerve stimulating drugs that were used in conjunction with it. However, the previously disappointing results with the chemotherapeutic agents alone I think speak rather loudly in favor of the microwave as the effective agent in the miprovement.

At the Institute of Ophthalmology, of the Presbyterian Hospital in New York, we began our experiments with the microwave about the same time that Dr. Clark did but with a completely different purpose in mind. Our objective was to use the microwave to reproduce the temperatures derived from foreign protein shock therapy and to use it in the form of a control in studies of the mechanism of foreign protein therapy on diseases of the eye. We began by animal experimentation, using anesthetized rabbits. Our object was primarily to find out if we could reproduce the intraocular temperatures of generalized fever; second, what the treatment doses would be to produce and sustain those fevers for the time interval required; and third, and, of course, most important, if there was any harmful effect on the tissues of the eye produced by the microwave therapy.

As the experiments were carried out, we found that it was very simple to produce temperatures from 103° to 105° F. in the vitreous chamber by the use of microwave radiation, the temperatures being taken by a thermocouple attached to the potentiometer in the routine fashion. The factors which I will give later, are the same as those we used in the treatment of the human eye. We then carried out a series of experiments in which we exposed the eyes of animals to a standard treatment of exposure for twenty minutes daily for five days under these same temperature factors. At the end of this period one series was sacrificed and immediate sections made: the second at a three-month interval, and the third at a six-month interval. The treatments used were higher than any that have been mentioned by either Dr. Clark or Dr. Thorpe because we were trying to reproduce the temperatures we experienced in sustained foreign protein therapy; therefore, we were using 60 per cent output instead of 20 to 30. There was, however, no damage shown in any ocular tissue in any of the animals immediately, at three months or six months thereafter. I think it is important to stress this because there have been a number of papers recently describing cataract formation due to microwave therapy. I think it is misleading because if you will notice carefully the temperatures producing the cataracts ranged from a low of 146 to an actual high of 158 degrees. That is beyond the therapeutic range and it is beyond the tolerance of a patient.

Following our animal experimentation clinical evaluation began in which we compared our results on patients treated with the microwave as opposed to those treated with foreign protein therapy. We have a series of 95 cases under each in which we confined ourselves to

iridocyclitis, retained cortex, and postoperative uveitis. Our standard dose was given with 60 per cent of the output, using the Director C, which is the angular director described by Dr. Thorpe, at a distance of four inches from the closed eyelid and perpendicular to it. The treatment was carried out every other day for twenty minutes at each treatment. In this series we can also report there were no harmful effects noted at or since that time in any of the patients treated. The subjective reactions of the patients were noted, and they varied from a very soothing sense of warmth during the treatment to a very marked and welcome relief of symptoms. The actual result, objectively checked on the patients, showed that it was not as good a reaction as we gain in the use of foreign protein therapy but, it did, in every case, reduce the expected course of the disease.

Dr. Clark's further studies may give us a great deal of interesting information, and certainly he has cast a ray of hope in a field badly in need of a new ray of hope. We will be most interested in his further reports.

BINOCULAR INDIRECT OPHTHALMOSCOPES

The Trotterscope and the Heedscope

JOSEPH M. DIXON, M.D.
BIRMINGHAM, ALA.

BY INVITATION

WHILE I was at the Massachusetts Eye and Ear Infirmary in Boston on the Heed Fellowship, Dr. Robert Trotter generously and informally demonstrated his partially constructed arrangement for binocular indirect ophthalmoscopy. Schepens[3,4] had shown that his method of fundus examination had certain advantages. It was Dr. Trotter's idea that the housing and rhomboid prism unit of a Bausch and Lomb Binocular Magnifier without magnifying lenses would satisfy the optical requirements. During the remainder of the Heed Fellowship many individuals* who observed the further development of this idea made valuable suggestions. Several constructed their own instruments, and the instruments vary with the maker. All of these instruments are variations of the 1861 Giraud-Teulon[1,2] binocular indirect ophthalmoscope modified by the attachment of an electric light to the double reflecting system.

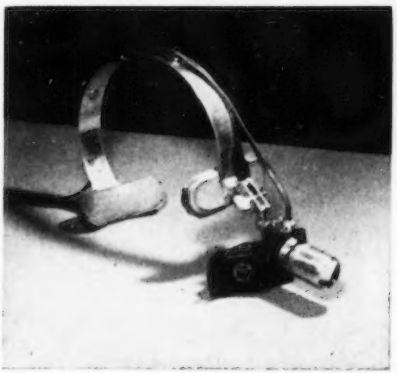
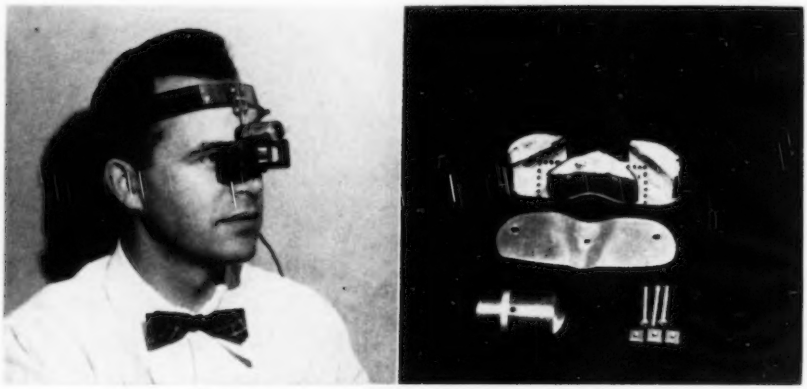


FIG. 1—Trotterscope, made by attaching rhomboid prism unit of Bausch and Lomb Binocular Magnifier to headband by swivel. Headlight attached by second swivel.

The only requirement for a binocular view of the indirect fundus image is that the two visual lines be brought near the axis of the light source. A pair of rhomboid prisms will do this by producing an artificially narrow P.D. of 15 to 20 mm.

From the department of ophthalmology, Northwestern University.
Heed Fellow in Ophthalmology.
Presented as *New Instruments* at the Fifty-Sixth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 14-19, 1951, Chicago, Ill.
*Dr. James Culver, Chicago; Drs. Graham Clark and Herbert Katzin, New York; Mr. Edward Vaupel, New York, and Dr. Ralph Burroughs, Birmingham.



A B

FIG. 2.—(A) Heedscope, model with four mirrors attached to plywood, assembled and fixed to housing of condensing lens of headlight. (B) Path of light from image to observer, and parts of Heedscope bolted together with aluminum instead of plywood.

A Trotterscope (fig. 1) may be made by attaching a small metal ball to the end of the shaft on the housing of the binocular magnifier and placing a pin through the shaft near the ball. The ball attaches the unit to a headband by a swivel, while another swivel over the pin supports the light.

A metal headband makes an excellent support. The National Headlight illuminating unit* with condenser can be regulated to furnish as much light as desired. Numerous types of transformers are satisfactory. Presbyopic examiners should wear their reading addition. Other examiners find that + 1.00 spheres make fusion easier for near.

In the Heedscope (fig. 2) four first-surface mirrors replace the rhomboid prisms, making it lighter in weight and inexpensive. The angle formed by the two central mirrors with each other is about 83 degrees, and the dimensions of the mirrors 20 by 37 mm., with 12 mm. separation of the mirrors on each side. These dimensions may vary reasonably. Alignment is made by observing a near object through the instrument and adjusting the wooden blocks holding the mirrors until fusion is comfortable. The blocks may then be fixed by bolts or glue.

The mirror unit is attached by a small bolt to the condenser housing of a National Headlight with the light tilted down. Inverting prisms can be used to make the indirect image upright, but the inverted field around the fundus image is annoying.

The two most useful condensing lenses are +14.00 and +20.00 diopters, about 50 mm. in diameter, available from local opticians.

This presentation is intended to make available simple and easily constructed equipment for binocular indirect ophthalmoscopy.

*National Electric Instrument Co., Elmhurst, N. Y.

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WHAT WE HAVE LEARNED ABOUT THE PHYSIOLOGY OF HEARING AS THE RESULT OF THE FENESTRATION OPERATION

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EVER since it was first observed that the creation of an opening within the vestibular labyrinth in ears deafened as a result of stapedial foot-plate ankylosis in otosclerosis results in improved hearing, it became quite obvious that whatever the physiology responsible for this phenomenon may be, the surgery is physiologically correct. Various theories were advanced to explain this physiologic phenomenon. Let us examine how these theories stood up in the face of the observations made following this surgery.

In pioneering the surgery for otosclerosis Holmgren[2] advanced the perilymph decompression theory in explanation of the hearing improvements following the fenestration operation. Sourdille[6] while subscribing to the perilymph decompression theory, argued also that in order to obtain practical useful hearing following fenestration in otosclerosis the maintenance of the incus in its original position after removing the head and neck of the malleus is absolutely essential.

In 1938[3] I advanced the theory that improved mobilization of perilymph and endolymph by air-borne sound is responsible for the improvements in hearing obtained following fenestration surgery; and in the May 1940 issue of the Archives of Otolaryngology[4] I made the following statement:

> The Lempert fenestration technic for permanent restoration of practical physiologic hearing in cases of otosclerosis is based on the theory that unimpeded mobility of the perilymph and endolymph for air-borne sound is essential to normal hearing by air conduction and that any impediment to such mobility results in deafness.

In order to determine which one of these theories is correct it became necessary to investigate them and determine, if possible, which one of these can be correlated best to the facts observed.

Békésy[1] has proved experimentally in cadaver material that increased perilymph pressure does not cause stapedial fixation. Wever, Lawrence, Meltzer and I[5] have demonstated in the rhesus monkey that increased perilymph pressure does not result in impaired cochlear nerve potentials. Furthermore, the fact that the creation of a fenestra in the cochlear portion of the labyrinth does not improve hearing in otosclerosis, plus the fact that only a fenestra made in the vestibular part improves the hearing, is sufficient proof that there is no basis for belief in the validity of the perilymph decompression theory. If increased perilymph pressure exists in otosclerosis and is responsible for the loss of hearing of air-borne sound, then the hearing improvement obtained with the newly created fenestra could not be maintained any appreciable length of time following the necessary sealing of that fenestra with the membranous tympanomeatal flap, which prevents the continuous decompression of increased perilymph pressure. Yet hearing improvements following the Lempert fenestra nov-ovalis operation have to date been continuously maintained for periods as long as 12 years.

Presented before the meeting of the Otosclerosis Study Group, Oct. 13, 1951, Chicago, Ill.

The fact that Sourdille's theory cannot be substantiated came to light when I developed the fenestra nov-ovalis technic whereby the incus is always removed without diminishing the degree of hearing improvement obtainable following fenestration when the incus was left in situ. However, failure to substantiate Holmgren's and Sourdille's theories did not necessarily prove that my theory of improved perilymph and endolymph mobilization by air-borne sound following fenestration surgery could be upheld as being any more logical or the reason for the improved hearing. Being fully cognizant of this fact I constantly studied experimentally both in lower animals and in the human the effect of various anatomic reconstructions of the middle and inner ear upon the physiology of hearing. I studied the behavior of hearing function following pathologic middle ear changes occurring either as a result of suppuration or otosclerosis. I also studied what happened following labyrinthine fenestration for clinical otosclerosis or following middle ear surgery for suppurative middle ear lesions, hoping thus to observe if either success or failure to improve the hearing could be correlated to my perilymph and endolymph mobilization theory. To anyone acquainted with the anatomy of the air-filled middle ear and fluid-filled internal ear it becomes quite obvious that since air-borne sound cannot penetrate the inner ear fluids without loss of energy, the middle ear is anatomically so constructed as to favor the channeling of sound energy from the tympanic membrane towards the basilar membrane by mobilizing the perilymph and endolymph within the lumen of the bony labyrinthine capsule.

The tympanic membrane with the ossicular chain attached to it forms the outer aspect of the middle ear channeling system for air-borne sound energy. The stapedial foot plate facing the perilymph and being firmly held within the vestibular oval window of the internal ear by the readily mobilizable annular ligament forms the innermost aspect of this channeling system. The cochlear endolymphatic labyrinth which houses the organ of Corti forms the scala media of the cochlear bony labyrinth. It is separated from the scala vestibuli by Reissner's membrane which forms the floor of the scala vestibuli, and from the scala tympani by the basilar membrane which forms the roof of the scala tympani, both of which are filled with perilymph. While the scala vestibuli communicates with the vestibule, the scala tympani is completely separated from the vestibular labyrinth. The ossicular chain thus channels sound energy across the vestibular side of the basilar membrane by mobilizing the inner ear fluids through the oval window. In order to facilitate the mobilization of these two labyrinthine fluid media and the channeling of air-borne sound energy across the basilar membrane through the two labyrinthine fluids the existence of an escape path on the side of the basilar membrane, opposite to the side where the pressure by air-borne sound is exerted, is essential. The flexible yielding membrane which seals the round window within the cochlear portion of the osseous labyrinth and which communicates with the scala tympani acts as the escape pathway for the displaced fluids. Air-borne sound pressure is also channeled from the tympanic membrane without the aid of the ossicular chain across the tympanic side of the basilar membrane by mobilizing with lesser intensity the inner ear fluids through the round window membrane which communicates with the scala tympani. The basilar membrane with the organ of Corti is thus put into a vibratory state (fig. 1).

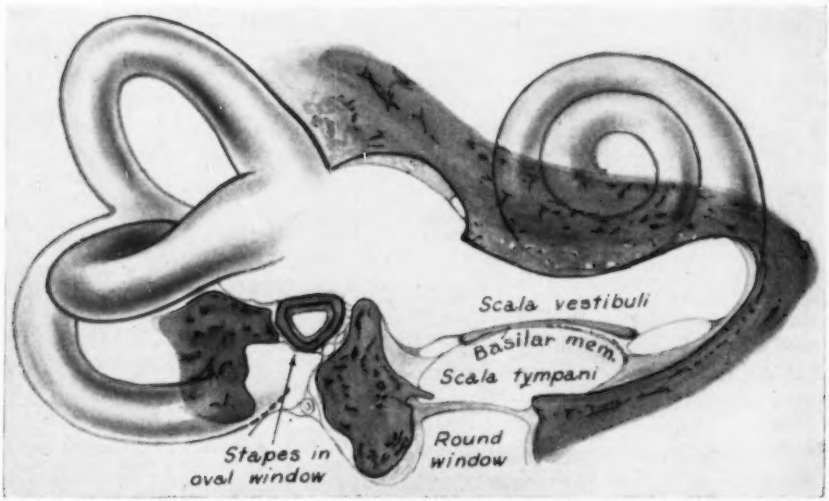

FIG. 1—Normally air-borne sound pressure is channeled across the vestibular side of the basilar membrane by mobilizing the perilymph and endolymph through the oval window which communicates with the scala vestibuli, and across the tympanic side of the basilar membrane by mobilizing the perilymph and endolymph through the round window membrane which communicates with the scala tympani, with the necessary phase difference in the pressure relationship to put the basilar membrane into a vibratory state.

By accepting this theory that a proper degree and type of mobilization of the basilar membrane through a system of channeling sound energy from the lighter particles of the air through the heavier particles of the inner ear fluids is necessary for normal hearing by air-conduction, it is not difficult to understand how any breakdown in this channeling system can impair the hearing for air-borne sound, whether the cause of this breakdown be the result of otosclerosis or middle ear infection. Clinically we know that impedance of the channeling function in the human ear can be overcome by increased intensity of sound providing the cochlear nerve function is not impaired.

As a result of observations made by Wever and Lawrence following an extensive study of cochlear response to air-borne sound in the cat under various forms of impedance created to the transmission of sound energy from the air through the labyrinthine fluids, they stated as follows:[7]

As it turns out the middle ear has the function of matching the impedance of the air to that of the cochlear fluid. If no such transformer system were present there would be a loss of 30 decibels in the transfer of energy from air to liquid. The drum and ossicle arrangement manages to overcome this to the extent of 28 decibels, and does so probably by the simple method of increasing the pressure at the foot-plate of the stapes through a concentration of force. This concentration comes about through the large forces acting on the drum being transferred through a chain of bones acting like a columella on the relatively small foot-plate of the stapes.

As a result of an experimental study in the rhesus monkey, by Wever, Lawrence, Meltzer and me, of the effects of the fenestration operation upon the breakdown of the middle ear system of channeling sound energy from the air to the basilar membrane, my perilymph and endolymph mobilization theory was confirmed. Wever and Lawrence[7] stated: "The fenestration operation as we have seen affects a remobilization of the cochlear fluid."

In the May 1940 issue of the Archives of Otolaryngology, I[4] stated as follows:

> If in a case of air conduction deafness due to impediment resulting from otosclerosis permanently free mobility of the perilymph and endolymph for air-borne sound could be surgicoanatomically re-established and the air conduction mechanism could be reconstructed to conform to the new surgical anatomy so as to enhance further the newly established mobility of these endolabyrinthine fluids for air-borne sound, permanent restoration of practical physiologic hearing should be obtained provided the hearing by bone conduction in the three conversational frequencies, 512, 1024 and 2048, is within the indicated limits of normal at the time of operation.

The observations which I have made as a result of experimental fenestration surgery in the human also appear to support my theory that the degree and type of improvement in hearing for air-borne sound following the fenestration operation is dependent directly upon the degree and type of improved mobilization of the perilymph and endolymph by air-borne sound energy resulting therefrom.

The successful restoration of hearing following a breakdown of the sound energy channeling system in the presence of unimpaired cochlear nerve function will depend upon our ability to repair this system or to substitute another channeling system surgically. The successfully performed fenestration operation, to my mind, does just this. It provides a new system for mobilizing effectively the perilymph and endolymph so that air-borne sound could be channeled across the basilar membrane.

Does the Lempert fenestra nov-ovalis technic as universally employed to date for the improvement of hearing in clinical otosclerosis provide the best available sound energy channeling system? It has been estimated experimentally by Wever and Lawrence that the normally functioning ossicular chain channels 30 times the pressure of the sound from the tympanic membrane to the perilymph and endolymph.

A newly created fenestra in the external semicircular canal following the fenestration operation channels approximately 30 to 100 times the sound pressure of the preoperated functionally impeded ossicular chain.

However, my clinical observations following labyrinthine fenestration in the human suggest that the degree of air-borne sound energy channeled by a newly created labyrinthine fenestra will depend upon how well the following three surgical objectives have been reached.

Objective No. 1

The primary objective of creating a new fenestra is to render the perilymph and endolymph freely mobilizable by normal air-borne sound so that the basilar membrane could be put into a vibratory state.

Extensive experimental fenestration surgery in the human has demonstrated beyond a shadow of a doubt that in order to mobilize the perilymph and endolymph by air-borne sound in the presence of stapedial footplate ankylosis a fenestra must be made only in the vestibular portion of the bony labyrinthine capsule. It has also been amply proved that the greatest amount of mobilization is obtainable when that fenestra is created within the external semicircular canal. It has also been demonstrated that such perilymph and endolymph remobilization is only obtainable when the round window membrane has remained normal and can act as an escape path to the sound pressure exerted in the scala vestibuli on the basilar membrane. When a new fenestra was created within the cochlear promontory instead of within the external semicircular canal, remobilization of the labyrinthine fluids was not accomplished. The reason a cochlear

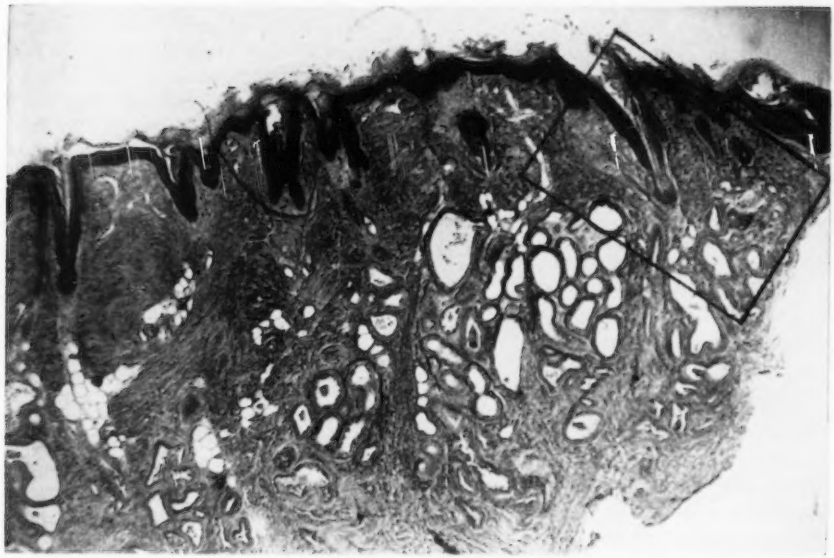

FIG. 2—Fresh skin flap showing the presence of hair follicles, subcutaneous fat and sebaceous glands.

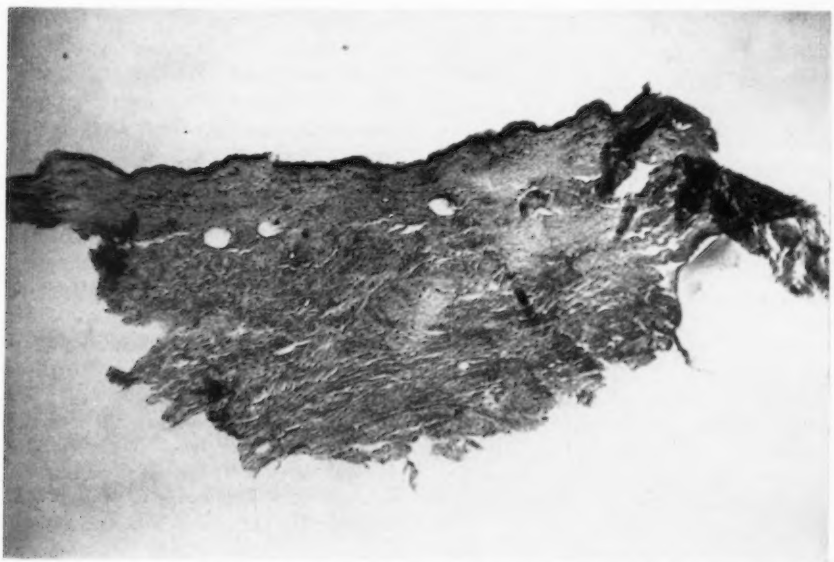

FIG. 3—Skin flap five years after placement. Atrophy of glandular structures has occurred and dense fibrosis is evident.

fenestra did not result in remobilization of the inner ear fluids was evidently the fact that in order to put the basilar membrane into a vibratory state the inner ear fluids have to be mobilized on both sides of the basilar membrane, i.e., through both the scala vestibuli and the scala tympani.

Objective No. 2

The second objective is to permit normally tolerated air-borne sound to do the mobilization of the perilymph and endolymph.

As a result of observations made by Lawrence, Wever, Meltzer and myself following an elaborate study of cochlear response to air-borne sound by the rhesus monkey under various forms of impedance artificially created within the middle ear sound channeling system, my mobilization theory was further confirmed. Wever and Lawrence stated as follows:[7]

> Alterations in the conductive system varying from the slightest changes in mass, stiffness, or friction to complete fixation or removal can produce an unlimited variety of audiometric pictures.

Clinical observation which I have made following fenestration surgery has demonstrated to me that the portion of the tympanomeatal membrane which is employed to cover and seal the fenestra gap, in order to permit the best possible mobilization of the perilymph and endolymph by sound energy, must be flexible and readily yielding. Experience has also shown that the thickness or thinness of that membrane has no influence on the mobilization of the perilymph and endolymph just as long as it remains flexible and yielding. However, experience has further shown that a thin membrane remains flexible for a much shorter postoperative time than a thick membrane. The reason for this, as observed during revisions, is that a thin membrane undergoes fibrosis and becomes stiffened much sooner than a thick membrane (figs. 2 and 3). It has been observed repeatedly that in the presence of a thin membrane the hearing improvement may drop 10 decibels in at least two of the vital speech frequencies, 512 and 1024, within a period of one year or 18 months and sometimes sooner, although the fenestra has remained permanently patent. When this occurs in an ear where the hearing has been restored to the 30 decibel level, which represents practical hearing, such a drop of 10 decibels means the difference between hearing and deafness. It is crucial. In a case where the hearing has risen to the 10 or 15 decibel level, such a drop still leaves the patient with practical hearing, though not as good as before.

In the presence of a thick flap, rich in subcutaneous soft tissue, such drops in hearing as a rule have not been observed, and patients with this type of flap have continued with their hearing at the original level for periods as long as 12 years. I am sure that many men doing this surgery can point to some of their earliest cases, which have maintained a high level of hearing improvement ever since, despite the fact that they covered the fenestra with the thickest portion of the flap because of their inability to cut a flap so that the thinnest portion would face the fenestra region. A thick vascular flexible flap keeps membrane facing the fenestra gap tuned longer for the low frequencies. Time changes the tuning quality of the flap sooner in the thin flap than in the thick flap. Further proof of the importance of the flexibility of the flap in the region of the fenestra for proper mobilization of perilymph and endolymph is the observation which I have made that in many cases the hearing postoperatively will maintain a high level of

practicability for a very long time in the presence of moisture within the postoperative mastoid cavity, and as soon as that moisture within the cavity disappears, or as soon as the cavity becomes epitheliated and dry, the hearing drops about 10 decibels. Unquestionably in the presence of a thin flap which gradually becomes fibrotic and stiffened, moisture in that cavity was most likely the factor which kept this flap flexible and enabled it to yield best to the pressure of sound for the mobilization of the perilymph and endolymph.

Patients with chronic middle ear infections hear better when the ear is discharging than when the ear is dry. In chronic suppurative middle ears the moisture renders the annular ligament more flexible and yielding to mobilization of the stapedial foot plate by air-borne sound. It is also not unusual to find, in patients appearing several years postoperatively for check-up of their hearing, that when tested before cleaning their postoperative cavity a drop of about 10 decibels in the speech frequencies will be noted. After cleaning the cavity and removing a film of dry secretion or cerumen covering the flap in the region of the fenestra, the audiometric reading taken then will show an improvement of 10 decibels as compared to the one taken before this crust had been removed. This crust rendered the flap less yielding to the pressure of air-borne sound and has interfered with the proper mobilization of the perilymph and endolymph. Many of us have operated upon very suitable cases of otosclerosis and following the operation the hearing was not improved, and many of us could not understand the reason for it and thought that perhaps the case, after all, was not a suitable one. However, it is my belief that in those cases some of the perilymphatic trabeculas have been injured and bleeding has taken place within the perilymph space after the flap has been placed in position. Fibrosis taking place within the perilymph space is sufficient to prevent improved mobilization of the perilymph and endolymph by air-borne sound, although not sufficient at times to result in a further impairment of hearing.

Objective No. 3

The third objective is to reconstruct the middle ear surgically so that it would permit the mobilization of the cochlear fluids by air-borne sound energy through both the new vestibular fenestra and Nature's cochlear fenestra under the most favorable phase relationship. Observations made in the rhesus monkey experiments by Wever, Lawrence, Meltzer and myself have prompted Wever and Lawrence[7] to state as follows:

> The action of the middle ear on the inner ear fluids is mechanical requiring that the other end of the system be free to move. The hair cells cannot be stimulated by the presence of sound pressure alone; there must be mobility of the fluids such that the mechanical effects of sound can actually move the basilar membrane back and forth. Sounds of equal energy entering at the two ends of the cochlear system can either double or completely cancel the movement of the hair cells depending upon the phase relation of the two stimulating sounds.

In order to channel sound energy across the basilar membrane and put the basilar membrane into a vibratory state by mobilizing the inner ear fluids following the fenestration operation, a phase or intensity difference in the mobilization of the labyrinthine fluids in front of the basilar membrane through the new oval window and in back of the basilar membrane through the round window must be created. The phase difference following fenestration is the time difference in sound mobilizing the fluids in front and in back of the basilar membrane. A hermetically sealed tympanic air space with an intact tympanic

membrane seems to play an important part in establishing the necessary and proper difference in phase or intensity relationship of sound pressure exerted on the vestibular and cochlear windows for the best obtainable mobilization of the perilymph and endolymph.

after the hearing had improved, the hearing inevitably receded to a level below practical and often to the preoperative level. As soon as the perforation in the tympanic membrane healed the hearing improvement again returned to the postoperative high level.

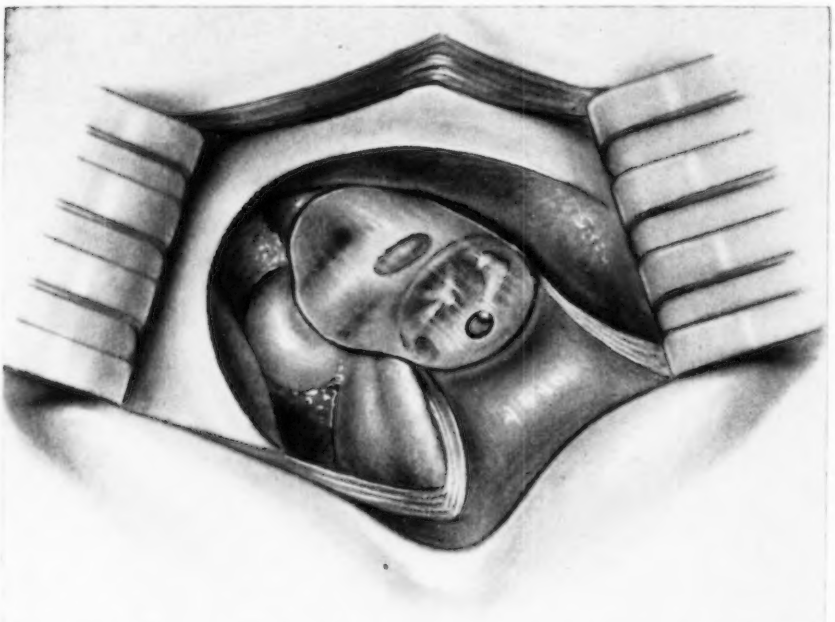

FIG. 4—Perforated tympanic membrane following the fenestration operation for clinical otosclerosis prevents improved hearing in operated ear until perforation heals.

I offer the following clinical observations following fenestration surgery in support of this.

Clinical Observation No. 1

Whenever the tympanic membrane was accidentally perforated during the performance of a fenestration operation the expected hearing improvement was inevitably delayed until the perforation healed. Whenever a hearing improvement was obtained following the fenestration operation and a perforation of the tympanic membrane had occurred

The explanation for this may well be that unlike the ossicular chain which increases the air-borne sound pressure on the oval window, the drum acts as a barrier and reduces the sound pressure against the round window. The difference in intensity at the two windows causes a movement of the fluids and puts the basilar membrane into a vibratory state. When the tympanic membrane also produces a phase difference between the two windows then the vibration of the basilar membrane is enhanced still further. Following fenestra-

tion there is a difference in intensity and also in phase in the two windows.

When the drum, during or following fenestration, is perforated the fluids are mobilized on both sides of the basilar membrane in equal intensity and phase, thus preventing the basilar membrane from being vibrated effectively by the sound energy and therefore a drop in hearing to the preoperative level results (fig. 4).

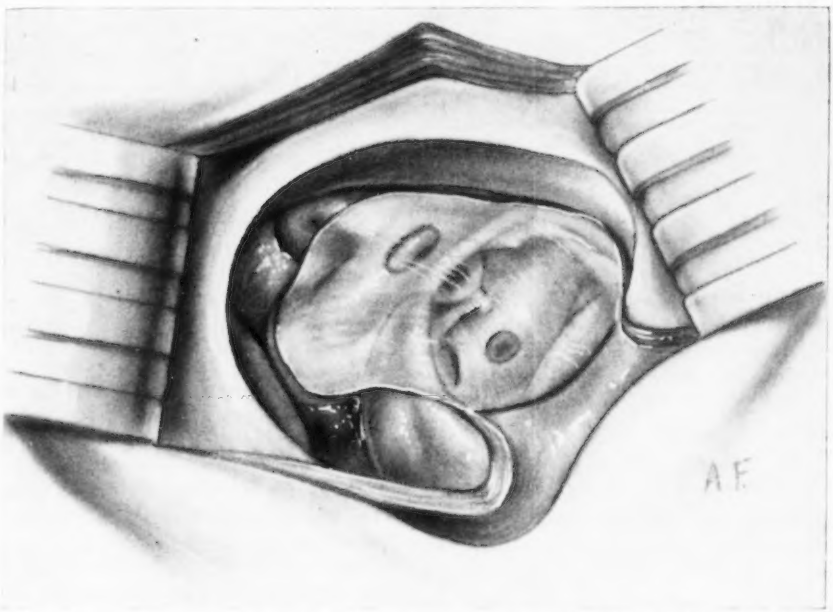

FIG. 5—When in the presence of stapedial fixation and a normal round window membrane in addition to creating a fenestra within the external semicircular canal a fenestra within the scala tympani of the cochlea is also created, the rightfully expected restoration of practical hearing is prevented.

Clinical Observation No. 2

In one of the experiments upon an otosclerotic ear with good cochlear nerve function, the tympanic membrane and ossicular chain were removed and two new fenestras were created; one in the external semicircular canal and the other in the scala tympani of the cochlear portion of the osseous labyrinth. Both new fenestras were covered and sealed with a split thickness skin graft. Paraffin mesh gauze was tightly packed into the postoperative mastoid cavity and the tympanic cavity, blocking both new windows to air-borne sound (fig. 5). Twelve days postoperatively the packing from the mastoid cavity, which was blocking the newly created fenestra in the surgical dome of the vestibule, was removed, thus freeing it for sound transmission to the scala vestibuli of the cochlea. The packing which was blocking the newly created window in the scala tympani of the cochlear promontory was not disturbed. At the end of three weeks postoperatively an improvement of hearing in the operated ear manifested itself which, however, was

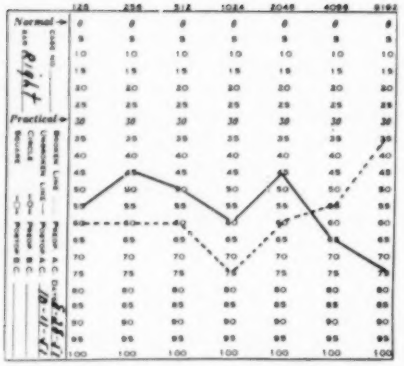

FIG. 6.—After the packing in front of external semicircular canal fenestra was removed. Packing in front of the newly created window in the scala tympani of the cochlear promontory not yet removed.

not of sufficient practical value to the patient (fig. 6).

This hearing improvement remained unchanged until six weeks postoperatively when I removed the packing held against the newly created fenestra within the cochlear promontory, which was blocking sound from entering into the scala tympani. Immediately following the removal of this packing the hearing dropped to the preoperative level (fig. 7).

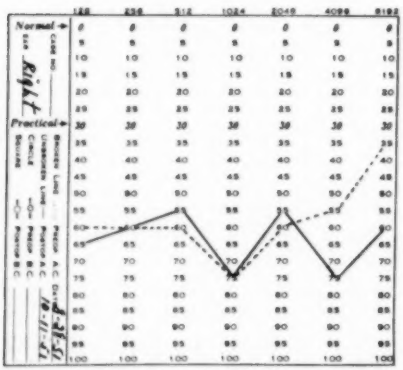

FIG. 7.—After the packing in front of both the new vestibular and the new cochlear window was removed.

Explanation. This experiment shows that the sound pressure on the cochlear side was greater when only the normal round window was permitted to function. As soon as sound was permitted to press also on the second cochlear window after removing the pack, whatever difference in phase between the scala tympani and scala vestibuli fluid movement existed was largely neutralized.

Clinical Observation No. 3

Further proof, and perhaps most important proof, that the improvement in hearing following fenestration is due to the mobilization of the perilymph and endolymph out of phase is the following:

When a fenestra is made in the external semicircular canal in the presence of a normally functioning ossicular chain, normal round window and normal intact drum, the hearing for air-borne sound becomes impaired.

In a patient with normal hearing in both ears who was suffering from distressing and incapacitating monaural tinnitus I performed a tympanosympathectomy for the relief of the tinnitus. This was accomplished through the external auditory canal by dislodging and everting the lower half of the tympanic membrane which was subsequently replaced into its normal position. Since this was accomplished without disturbing the ossicular chain the hearing loss following this surgical intervention was trivial (fig. 8).

The tinnitus, however, was not relieved. Twenty-six months later, at the insistence of the patient that something must be done to relieve her of this unbearable tinnitus, I made a fenestra in her external semicircular canal without disturbing her ossicular chain and tympanic membrane. My decision to open the external semicircular canal for the relief of her tinnitus was based upon my

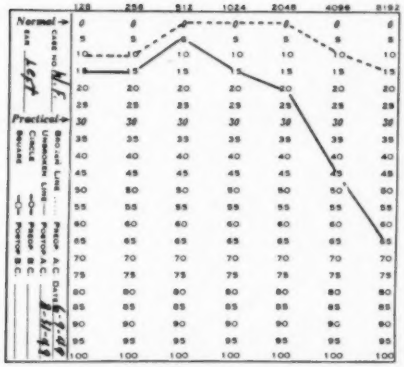

FIG. 8—Hearing loss following tympanosympathectomy for relief of tinnitus in an otherwise normal ear.

observation that following the fenestration operation for clinical otosclerosis, tinnitus in the operated ear disappears in about 90 per cent of the ears. Furthermore, the fact that the tinnitus in these cases returned whenever osteogenetic closure of the fenestra took place was strongly suggestive of the possibility of relieving tinnitus in a nondeafened ear as well, creating a fenestra in the external semicircular canal.

Despite the fact that this patient's external semicircular canal was opened and sealed with the tympanomeatal flap, which was created without removing the innermost bony margins of the posterior and superoposterior walls of the external auditory canal and without disturbing the tympanic membrane and ossicular chain, a considerable recession in the preoperative hearing level resulted postoperatively (fig. 9).

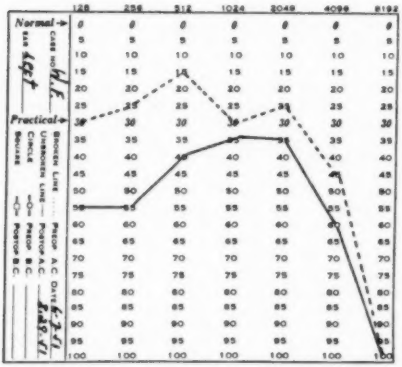

FIG. 9—Hearing loss following the creation of a fenestra in external semicircular canal without disturbing any of the middle ear anatomy for the relief of tinnitus in an otherwise normal ear.

The tinnitus unfortunately was not relieved.

Explanation. This hearing level recession may be explained by the fact that sound pressure exerted on the oval window by the normal ossicular chain is being dissipated by way of the new window and is no longer being forced entirely in the direction of the basilar membrane (fig. 10).

CONCLUSION

In conclusion I would like to state that what still remains unknown about the physiology of hearing will some day fill volumes in the literature. I also am cognizant of the fact that all I know about the physiology of hearing could most likely be inscribed upon the head of a pin. However, the clinical observations which can be repeatedly made following fenestration of the labyrinth in the human unquestionably support my theory that the hearing improvements following fenestration are the result of remobilization of the perilymph and endolymph by air-borne sound with a difference in intensity and phase in the two windows.

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FIG. 10—The creation of a fenestra within the external semicircular canal of a fairly normal hearing ear without disturbing the normal ossicular chain and normal tympanic membrane results in impaired hearing for air-borne sound.

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BONE CONDUCTION ADVANCES FOLLOWING FENESTRATION SURGERY

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BY INVITATION

SEVERAL investigators have reported that improvement in bone conduction acuity follows fenestration surgery. However, the details of the change have been only partially described and its implications have gone largely unnoticed. Therefore, it is pertinent to examine the topic critically and inquire into its practical significance.

I

As a preliminary point, we must avoid a very widespread misconception regarding the bone conduction audiogram. The bone curve is not, in most cases of conductive hearing impairment, a completely accurate picture of cochlear function. The mechanical abnormalities which produce the hearing loss modify not only the air but also the bone thresholds. The effects on the bone curves vary with the nature of the lesion and they are of limited magnitude. It is the cochlear response which determines the general position of the bone curve on the audiogram chart. The influence of the conductive lesion is to add a distinctive distortion to a bone curve whose basic pattern is determined by cochlear function. Stapes fixation, for example, produces a notch-shaped distortion which superimposes itself upon the patient's pattern of cochlear reserve.[1] Hence, clinical otosclerosis without secondary nerve involvement typically yields an audiogram wherein the bone curve lies a little below the zero line. Cochlear function is here normal, but the curve does not reveal the fact directly because of the superimposed notching. Similarly, otosclerosis with secondary nerve involvement appears from the bone audiogram to have somewhat greater sensory-neural impairment than is actually the case. Thus, we have in otosclerosis an excellent example of the fact that the bone audiogram is the composite product of two factors. The first, which has commonly been accepted as the only factor, is cochlear function. The second is the purely mechanical modification of response introduced by an appropriate conductive lesion. Naturally, where there is no conductive impairment, the second factor is absent.

The aspect of the foregoing principle which is important to us here may be stated as follows: surgical reorganization of the conductive mechanism may alter bone response without changing the patient's underlying cochlear sensitivity. Thus, it would be erroneous to assume that postsurgical improvement in bone thresholds is evidence that the operation restores neurosensory function. This improvement is merely a by-product, but the fact that it can be demonstrated gives us knowledge we may use to evaluate more precisely the cochlear reserves of patients with clinical otosclerosis.

II

McConnell recently completed a study which helps define the mechanical effects which fenestration surgery has upon bone conduction responses.[3] His

From the School of Speech and the Department of Otolaryngology, Northwestern University.
Presented before the Otosclerosis Study Group, Oct. 13, 1951, Chicago, Ill.

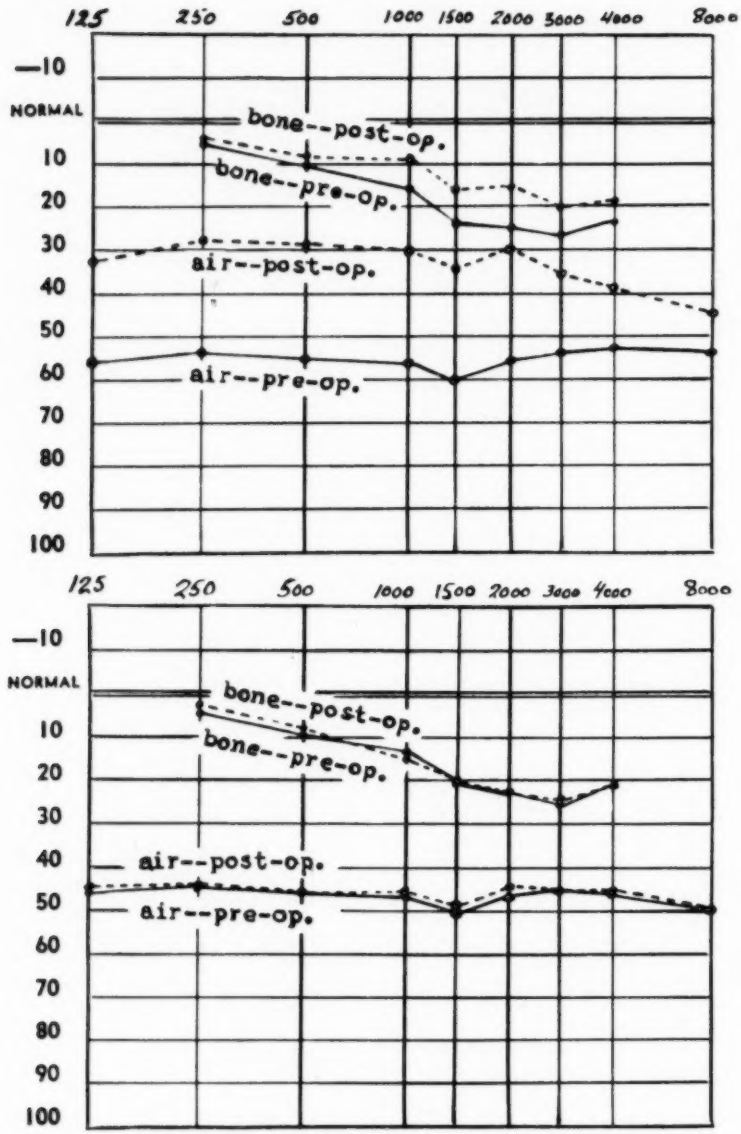

FIG. 1—Comparison of average preoperative and postoperative thresholds for air and bone on fifty-eight subjects. Upper chart reports data for operated ears and lower chart for unoperated ears. (After McConnell.)

basic procedure was simple. A consecutive series of 58 patients selected for fenestration surgery by Shambaugh and his associates was tested twice. One test was given to each patient two weeks before surgery and the other about fourteen weeks after operation. These tests were given in addition to those obtained by the surgeons for the purpose of determining each patient's suitability for the operation. All pure tone explorations were made with a commercial audiometer. Special precautions were taken to assure that the instrument was in adequate calibration and that it remained stable throughout the time span of the investigation.

The effect of surgery was evaluated by statistical analysis of the findings for ears subjected to surgery, while the threshold responses for the unoperated ears constituted control data. The configuration of these latter data demonstrated conclusively that technic and equipment were highly stable. Thus, the results for operated ears could be accepted as demonstrating clearly the effect of fenestration surgery on both air conduction and bone conduction thresholds.

III

We may now examine McConnell's findings.

As demonstrated by figure 1, which summarizes results in the form of audiograms, the fenestration operation produced marked improvement in the air conduction thresholds of the operated ears, while thresholds for the unoperated ears remained essentially unchanged. The average air gain in the operated ears was approximately 25 db. for frequencies from 125 through 2,000 c.p.s. The gain became progressively less above 2,000 c.p.s., being approximately 17 db. at 3,000; 13 db. at 4,000; and 9 db. at 8,000 c.p.s. Although individual variations were substantial, a fairly high interdependence was found between air gains at adjacent frequencies. Moreover, the average gain was largely dependent upon degree of preoperative conductive impairment. All these findings were to be expected. They demonstrate that the results for air conduction which McConnell obtained were typical of those yielded by patients selected for fenestration surgery.

Turning to the results on bone conduction, one notes in figure 1 that no appreciable shift occurred in the bone thresholds of the unoperated ears. In contrast, operated ears revealed a group pattern in which only a slight shift occurred for tones below 1,000 c.p.s., but a marked improvement took place throughout the range from 1,000 through 4,000 c.p.s. Statistical analysis demonstrated conclusively that the latter was attributable to surgery.

Figure 2 gives a more detailed picture of the postoperative changes in bone thresholds. The significant gains for operated ears were as follows: 6.5 db. at 1,000 c.p.s.; 7.4 at 1,500; 8.5 at 2,000; 6.4 at 3,000; and 4.2 at 4,000 c.p.s.

Individual patients deviated substantially from the foregoing pattern, so that the results must be viewed as only a group trend. One must conclude that factors as yet unknown were at work. When these factors are discovered through future research, they should help the surgeon to evaluate individual patients.

In the meantime, McConnell's results are important in demonstrating that the mechanical influences of fenestration surgery affect bone thresholds to a measurable degree.

IV

The change which McConnell observed was postsurgical improvement in

bone conduction thresholds. This shift is opposite in direction to the mechanical (noncochlear) depression in bone sensitivity caused by stapes fixation. Thus, the operation tends to reverse the distortion in the bone audiogram which stapes fixation imposes.

There is no *a priori* reason why this reversal should be complete. The fenestrated ear is not an ear restored to the physical state it possessed before stapes fixation developed. One therefore wonders whether a new pattern of mechanical distortion appears in postfenestration bone curves, so that even here bone thresholds are not perfect indications of cochlear function.

One may explore this possibility through further analysis of McConnell's data. Here, the results on postoperative air-bone gaps supply information of importance. The average magnitude of the separation between air and bone curves differed with frequency. Considering data for the entire group of patients, the average difference between air and bone thresholds was 24.4 db. at 250 c.p.s.; 20.8 at 500; 21.2 at 1,000; 18.3 at 1,500; 14.4 at 2,000; 15.9 at 3,000; and 20.8 db. at 4,000 c.p.s.

One can use these figures in arriving at an estimate of the amount by which fenestration surgery failed to reverse completely the preoperative shift in bone sensitivity due to stapes fixation. The process is simple. One need merely find a way of estimating the true size of the conductive deficit produced at each frequency by surgical disruption of the ossicular system. One then subtracts

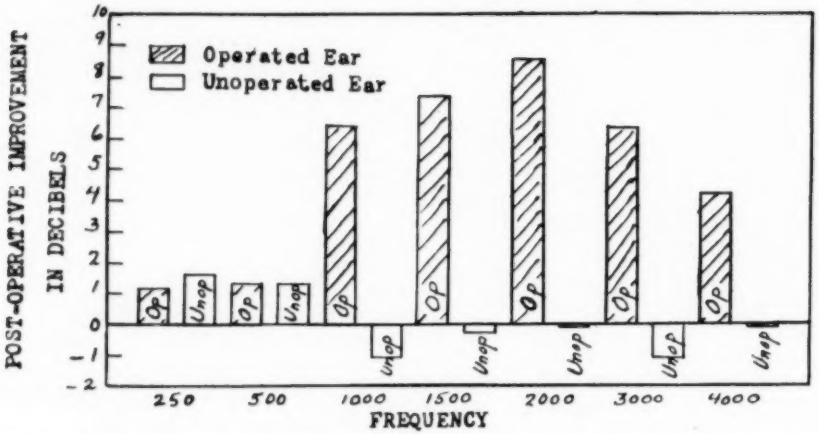

FIG. 2.—Comparison in mean improvement in bone conduction sensitivity of operated and unoperated ears of fifty-eight subjects. (After McConnell.)

from this value the postoperative air-bone gap at that frequency. The difference is the amount by which the air-bone gap fails to give an accurate indication of the conductive deficit. In other words, the difference indicates that portion of the original mechanical shift in bone response which fenestration has not erased. If no difference appears, one may accept the bone thresholds as unmodified indications of cochlear function.

V

An estimate of the conductive deficit due to ossicular disruption can be obtained by analyzing a scattergram show-

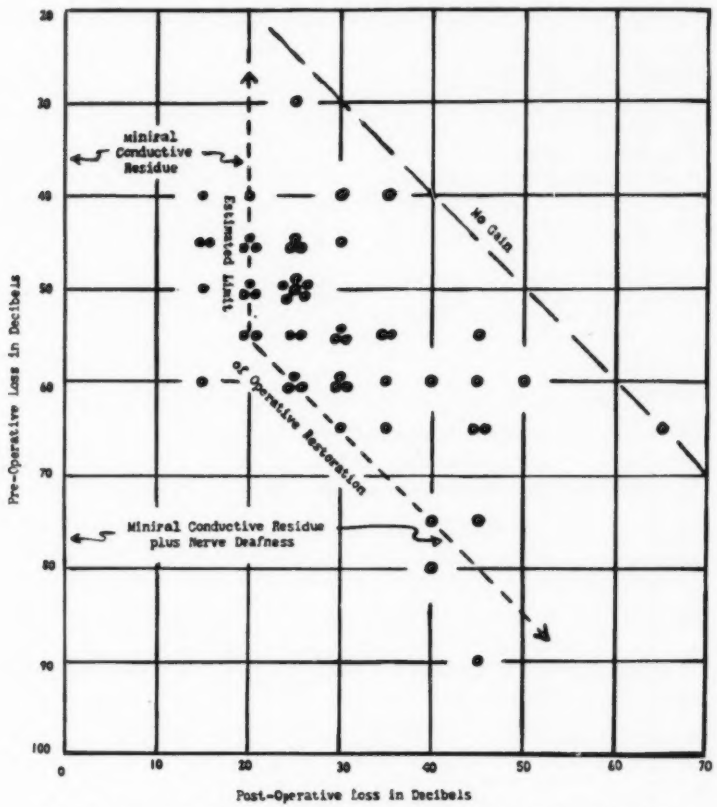

FIG. 3—Relation of preoperative and postoperative thresholds for air conduction at 2,000 c.p.s. (Plotted from McConnell data.)

ing distribution of air conduction thresholds. This method was used by Davis and Walsh[2] when they computed from speech thresholds the conductive deficit produced by surgical disruption of the ossicles. They reached the conclusion that this deficit is 20 db.

Figure 3 is typical of results one obtains when one treats the McConnell data on a single frequency in the same manner. Here, preoperative air thresholds are plotted against postoperative air thresholds. The results are not randomly distributed but fall into a definite pattern. The data shown in figure 3 are for 2,000 c.p.s. Observe that to the left there is a boundary labelled as the "Estimated Limit of Operative Restoration," beyond which very few postoperative scores fall. This limit was drawn in arbitrarily because the majority of scores lie along it or bunch close to it on the right. In other words, one assumes the underlying principles postulated by Davis and Walsh and then the boundary is located as required by these principles and the data at hand. The vertical portion of the boundary indicates the approximate size of the conductive deficit due to ossicular disruption. In figure

3 this vertical portion is 20 db. to the right of zero on the postoperative scale. One therefore estimates that, postoperatively, the minimal conductive deficit is 20 db. at 2,000 c.p.s. Moreover, the point on the preoperative scale at which the boundary breaks and begins to diagonal to the right is an indication of the maximal conductive impairment which stapes fixation produces at the frequency under study. McConnell's data lead to the belief that this maximum is 55 db. for 2,000 c.p.s.

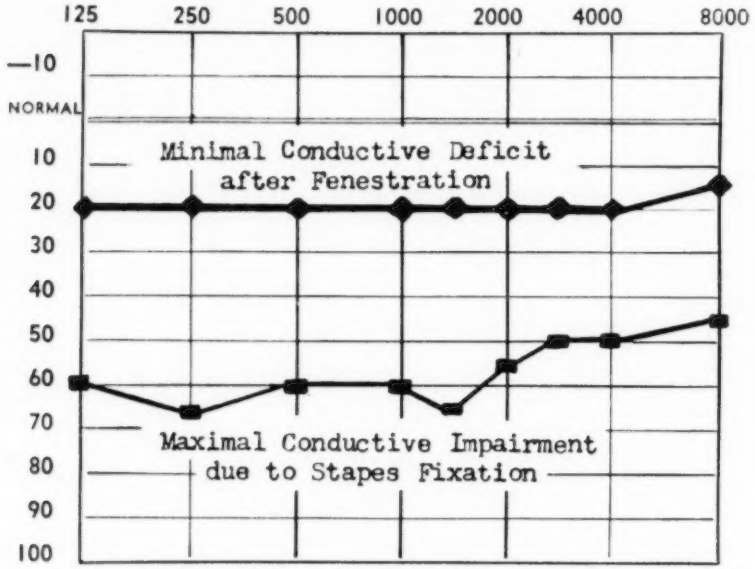

FIG. 4—Estimated limiting values of conductive hearing loss found in clinical otosclerosis. Any additional loss due to nerve involvement summates with conductive loss.

Similar analysis was made of the scores for each frequency from 125 through 8,000 c.p.s. The results are summarized in figure 4, which is in the form of an audiogram. The lower curve plots the maximal conductive impairment to be expected preoperatively at each frequency. This curve here requires only passing attention, since the main interest for the moment is the size of the postoperative conductive deficit. The latter is shown by the upper curve on the chart. This curve is the estimate of the minimal conductive deficit to be expected after surgery. The reader will observe that this deficit appears constant from one frequency to another. The findings are interpreted to mean that, for the group McConnell studied, the minimal postoperative conductive deficit due to ossicular disruption was approximately 20 db. across the frequency range from 125 through 4,000 c.p.s. but only 15 db. at 8,000 c.p.s.

The fundamental consideration in the foregoing discussion is that one may, on the basis of the distribution of air conduction thresholds alone, estimate the minimal conductive deficit which, after surgery, typifies the group one is studying.

It is such an estimate, derived from McConnell's data, which may be compared with the air-bone gaps that his group revealed in order to judge whether or not the postoperative bone thresholds were undistorted indications of cochlear function. As will be recalled, the size of the postoperative air-bone gap varied from one frequency to another across the band from 250 through 4,000 c.p.s., which was the range McConnell explored by bone audiometry. Now, remembering that the actual conductive deficit was estimated to be about 20 db. for these frequencies, the amount by which the air-bone gap at any frequency deviated from 20 db. may be interpreted as indicating the displacement in bone sensitivity which remained at that frequency after surgery. Pursuing the foregoing assumption, one needs merely compute the difference involved in order to estimate the effect of mechanical influences on the postsurgical bone audiogram. These differences, as derived from McConnell's data, were: -4.4 db. at 250 c.p.s.; -0.8 at 500; -1.2 at 1,000; 1.7 at 1,500; 5.6 at 2,000; 4.1 at 3,000; and -0.8 db. at 4,000 c.p.s. These values appear to be a reasonable indication of the amount by which the bone responses of McConnell's subjects remained mechanically modified after surgery. One concludes that, on the average, bone thresholds failed by these amounts to give a true picture of cochlear function.

VI

It is a simple matter to compute from the foregoing information the approximate size of the original "otosclerotic notch"; that is, of the preoperative displacement of the bone curve due to the mechanical influences of stapes fixation. The procedure is shown graphically in figure 5. One first draws curve A, which is derived from the gains in bone sensitivity which McConnell found were

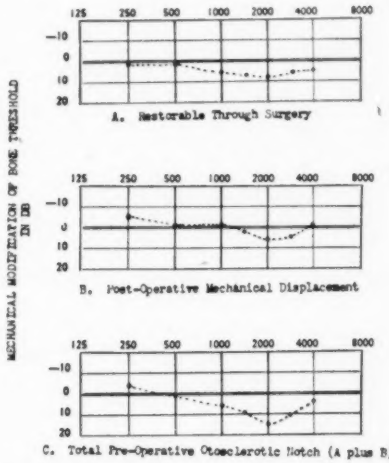

FIG. 5—Calculation of the magnitude of the otosclerotic notch (curve C). Derived by combining the bone sensitivity restorable through surgery (curve A) with the postoperative mechanical displacement (curve B), which was estimated from the air conduction responses of fifty-eight patients who underwent surgery.

produced by surgery. The curve is drawn below the reference, or zero, line because we are at the moment imagining a preoperative condition; that is, we are viewing the fraction of the mechanical depression which subsequent surgery was observed to reverse. One next draws curve B, which is the unrestored residue computed in the manner described above. This curve represents the mechanical distortion in bone conduction sensitivity which remained after fenestration. Finally, curves A and B are combined to yield curve C, which is the resulting estimate of the original "otosclerotic notch." In other words, since curve C is the sum of the observed restoration plus the postoperative residue, it is the total mechanical depression in the bone audiogram which existed prior to surgery. It is the average amount by which bone measurements gave a false picture of cochlear function.

Expressed numerically, such a procedure gives the following values for the preoperative "otosclerotic notch," when these values are computed for the fifty-eight patients under consideration. Bone sensitivity appears to have been mechanically improved 3.2 db. at 250 c.p.s. and mechanically depressed 0.6 db. at 500; 5.3 at 1,000; 14.1 at 2,000; and 3.4 db. at 4,000 c.p.s. It is interesting that Carhart,[1] in his original rough estimate of notch size, postulated no shift at 250 c.p.s.; 5 db. depression at 500; 10 at 1,000; 15 at 2,000; and 5 db. at 4,000 c.p.s. The discrepancies between the present computation and Carhart's original estimate are not large. The data at hand lead to the belief that the original postulation was not seriously in error and that, on the average, the "otosclerotic notch" may be assumed to have approximately the shape heretofore described. There will, of course, be individual variations in clinical records, and the significance of these variations needs further study. However, when one remembers that an audiometer measures hearing loss in 5 db. steps, a person having a true shift which satisfies the new estimate will yield a bone audiogram which agrees precisely with the original postulation. One concludes that we have at our disposal an average set of values which apply reasonably well for most cases of clinical otosclerosis. When a preoperative bone audiogram is corrected for this amount of mechanical distortion, the result usually should be an improved picture of the otosclerotic patient's cochlear function.

VII

We come now to the practical application of the foregoing concepts.

Here one must first introduce an additional fact. McConnell's data agree with those of Davis and Walsh in demonstrating that the majority of good surgical results did not quite achieve the minimal conductive residue of 20 db. An additional residue of about 6 db., the so-called "surgical deficit," was found repeatedly. Thus, the typical case showed, after operation, a conductive residue of approximately 25 db.

One may, as Shambaugh[4] has done, accept such a 25 db. residue as the most probable outcome of successful surgery. This assumption allows one to predict from preoperative records the anticipated postoperative audiogram for air; that is, the configuration of effective hearing expected from surgery. The steps are two. The "otosclerotic notch" is first subtracted from the bone curve obtained before operation. As mentioned, this correction gives an approximate estimate of the patient's cochlear reserve. Next, the postoperative hearing for air is predicted by drawing a new audiogram 25 db. below the corrected estimate of cochlear reserve. Shambaugh has demonstrated that such predictions agree well with clinical experience.

The fundamental point underlying this whole discussion is that allowance must be made for the fact that the bone conduction audiogram gives a distorted picture of the cochlear function possessed by an otosclerotic patient. Thresholds obtained before operation incorporate the "otosclerotic notch" and create the false impression of greater reduction of cochlear reserve than is actually the case. Thresholds obtained after operation incorporate a lesser mechanical distortion, but even here the bone measurements are not fully accurate indications of neurosensory function. However, as with the preoperative thresholds, appropriate allowance can be made for this mechanical influence and a reasonable postoperative estimate of cochlear reserve thus obtained. Among other things, such postoperative estimates, based on tests given at appropriate intervals, may be compared with

the estimate derived from the preoperative records. The stability of a patient's cochlear function may thus be judged with greater accuracy and long term changes in hearing may be followed with greater confidence.

Thus, when evaluating the hearing impairment resulting from clinical otosclerosis, as when dealing with other aural lesions, the otologist who uses insight in carrying out bone conduction audiometry gains helpful information. In the past, we have tended to emphasize the general level of bone thresholds rather than the details of audiometric configuration. This practice has resulted in part from the fact that mechanically induced shifts in bone response have been imperfectly appreciated. In consequence, changes in bone conduction threshold have sometimes been interpreted as modifications in cochlear function under circumstances where we now know these changes are purely mechanical by-products. Since mechanical shifts of this type are an integral part of the otosclerotic's auditory configuration, both preoperatively and postoperatively, we are in a position to apply a new level of interpretation to the clinical records of such cases. Only further research will reveal the full nature of such interpretation, but already enough has been learned to help the clinician make fuller use of his bone conduction measurements.

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NEUROANATOMY AND NEUROPHYSIOLOGY IN THE COCHLEA

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ST. LOUIS, MO.

BY INVITATION

THE HEARING loss in clinical otosclerosis is due primarily to impairment of the sound-conducting apparatus of the middle and inner ears. For its understanding the physical principles of acoustics are essential. The problem is one of biophysics. The hearing loss of nerve deafness, however, is a failure of the excitation of nerve fibers by specialized sensory cells. The problem is more a problem of neurophysiology than of bio-acoustics. Nerve deafness frequently complicates clinical otosclerosis, and some authorities believe that it may be a true part of the full otosclerotic syndrome in the sense that the physiologic failure and the pathologic alterations of the sense organ are caused, directly or indirectly, by the otosclerotic process. It is appropriate, therefore, to take you behind the scenes into a laboratory of physiology and review with you some of our present knowledge of the excitatory process and to theorize a little on the basis of some recent experiments. You will at least learn what kind of problems neurophysiologists are interested in.

THE INNERVATION OF THE ORGAN OF CORTI

Good physiologic theory requires accurate anatomic knowledge as a foundation. The relation of the nerve fibers to the hair cells is fundamental to any theory of the excitation of the auditory nerve as well as to auditory theory in general. This point has been studied carefully by Retzius, Cajal, Kolleker, Held and Lorente de Nó among others, and very recently by my colleague, Dr. Fernández. Dr. Fernández' work has been done entirely on guinea pigs, which is appropriate because our physiologic studies, for which his description is the basis, are also done on guinea pigs. It is a reasonable assumption, however, that the general organization of the innervation of the ear is similar in all mammals. Dr. Fernández has kindly given me permission to summarize for you his conclusions based on his own and preceding work. (The full description is now in press.[4])

The innervation of both the internal and the external hair cells is complex. In each case there are two classes of nerve fibers, the "radial" and the "spiral."

The radial fibers run directly outward from the spiral lamina and each fiber innervates one, two or, at most, three hair cells. The radial fibers form the chief nerve supply of the inner hair cells but they are in a minority in the supply of the external hair cells. [The radial fibers to the external cells were first described by Held as the "unbranched" fibers. The cell bodies of the radial fibers lie in the spiral ganglion of Corti, and all of them, as we would expect, are afferent sensory fibers.]

The spiral fibers, on the other hand, run for considerable distances, often several millimeters, in *both* directions along the organ of Corti. They give off many branches and each fiber thus innervates many sensory cells, although a given fiber does not innervate every hair cell along its course. A branch to every second or third cell is more typical. This type of innervation is predominant for the external hair cells. These spiral fibers form the well-known "external spiral bundles."

From Central Institute for the Deaf.

This work was carried out under Contract N6onr-272 between the Office of Naval Research and Central Institute for the Deaf.

Presented as "Cochlear Potentials, the Excitatory Process and Nerve Deafness" before the Otosclerosis Study Group, Oct. 13, 1951, Chicago, Ill.

A given external hair cell may be innervated by more than one spiral fiber. A given inner hair cell may receive endings from sometimes one, usually two or, at most, three radial fibers. In addition some, at least, of the inner hair cells also receive endings from spiral fibers, and external hair cells receive some radial fibers in addition to their spiral fibers.

The spiral fibers to the external hair cells are afferent, with cell bodies in the spiral ganglion. The spiral fibers to the internal hair cells, however, seem to be *efferent* fibers. Their cell bodies lie in the medulla oblongata in the superior olivary complex of the *opposite* side. They run from this nucleus in the [recently described[6]] tract of Rasmussen, and form a compact bundle in the auditory nerve. This bundle is clearly marked as it runs along the basal turn, forming the intraganglionic spiral bundle.

Dr. Fernández has identified this bundle as the continuation of the tract of Rasmussen, and has found that these fibers give off branches that terminate on internal hair cells. This spiral bundle and the fibers of the tract of Rasmussen do not degenerate, like the other fibers of the auditory nerve, when the organ of Corti is destroyed by severe acoustic trauma.

We have then, a double innervation, by diffuse spiral and by discrete radial fibers, for both internal and external hair cells; but the internal hair cells seem to have an *efferent* as well as an afferent supply.

We have not yet been able to assign separate functions to these various classes of fibers, but we must reckon with them in our theories of the neurophysiology of hearing.

EXCITATION IN SENSE ORGANS

We have no sure knowledge of the exact mode of action of *any* sensory cell in setting up nerve impulses. The function of specialized sensory cells in general is to make the nerve fibers more easily excited, by some particular form of energy such as light, temperature, specific chemical composition or mechanical deformation. The cochlea and the labyrinth are both in the latter class together with touch, pressure and muscle senses. Some stimuli, including mechanical deformation, if they are strong, can excite nerve fibers directly although the nerve fibers may often be injured in the process. Nerve fibers are also excitable by electricity.

Theories of the action of sense organs have tended to invoke these known excitatory effects on the nerve fibers as analog of the action of the sensory cells. The latter have often been considered as nerve cells that have specialized in the direction of some particular form of sensitivity that is represented to a lesser degree in all nerve cells.

The final stage of excitation of a nerve fiber is generally agreed to be electrical depolarization. The question is how the sensory cell that is attached to the nerve fiber causes this depolarization. The problem would be partly explained or at least pushed one step back if an electrical potential generated by the sense cell were demonstrated, because an electrical current following in the proper direction is a simple and obvious way of producing an electrical polarization or depolarization.

A very slight depolarization would suffice for a very excitable fiber, because it initiates an autocatalytic reaction that tends to produce a further depolarization. This *local excitatory process* is balanced against a stabilizing or recovery process. The critical event is the generation of a local excitatory process that covers a large enough area. Then the "threshold" is reached for a full-blown chain reaction, a self-propagating wave of depolarization, which is the nerve impulse. The precise time relations of a stimulus and the rates of build-up and decay of the local nonpropagated excitatory process are critical in determining its effectiveness.

Such local excitatory processes, which may outlast the original stimulus and which, in principle at least, can sum with one another if set up in neighboring parts of the same cell, have been demonstrated at the motor endplates of striated muscle and at the synapses of the central nervous system.

It is a tempting speculation that the significance of the multiple connections of several hair cells to a single nerve fiber may be to allow the summation of several weak excitatory processes in different hair cells so that they can *combine* to set up an impulse. Here may be an explanation of the extraordinary sensitivity of the ear to faint sounds. It may answer a very difficult question posed by De Vries[3] as to how the ear can be so sensitive to sound and still not be continually excited by thermal energy and the Brownian movement of its molecules.

THE COCHLEAR MICROPHONIC

The most popular theory of excitation of the auditory nerve invokes the well-known cochlear microphonic (Wever and Bray effect) as the intermediate mechanism. The latter is an electrical potential generated in the right place, namely in the organ of Corti. It does not seem to be the electrical sign of the local excitatory process in the nerve, however, for the latter would be in one direction only, like an action potential, and the microphonic is alternately positive and negative, following the pattern of the mechanical movement of the basilar membrane. If the microphonic plays a part in excitation it must represent an earlier step, the step which initiates the local excitatory process. Some of us have believed that the microphonic is merely an incidental and purely passive transformation of some of the mechanical energy of the stimulus into electricity. Békésy has just shown,[1] however, that apparently the tissues of the cochlear partition contribute at least part of the energy of the microphonic from their metabolic processes. If he is right, as he usually is, the case for the microphonic as the process that initiates the excitatory process becomes stronger. The question is further complicated by evidence that there are at least two kinds of microphonic, one of which is very dependent on an adequate supply of oxygen while the other continues for hours after the death of the animal. Beyond this the nature and origin of the cochlear microphonic are still completely obscure.

THE SUMMATING POTENTIAL

A little over a year ago we[2] described a third electrical potential in the cochlea, distinct from the microphonic and also from the "spike" of the action potential. The new potential begins *before* the spike. Unlike the microphonic it is always in one direction, and it has the important property of outlasting a single sound wave at the higher frequencies (i.e., above 2,000 c.p.s.). It seems to be generated in the organ of Corti. It is demonstrated by inserting very small electrodes into the scala vestibuli and scala tympani. Dr. Fernández is responsible for the beautiful surgical technic which involves "fenestrating" the cochlea of the guinea pig with as many as six different holes at once ranging in diameter from 15 microns to 25 microns. The electrical outputs from these electrodes are combined in such a way as to cancel out either the action potential or the cochlear microphonic. In this way the new (and somewhat weaker) "third potential" was studied. It seemed to have the properties predicted for the kind of local excitatory process described in an earlier section.

Unfortunately further work has complicated instead of clarified our picture. We now believe, thanks to the experimental skill and penetrating theoretical analysis of our new collaborator, Dr. Tasaki, that at least part of the "sum-

mating potential," as we called this early electrical wave, is really the action potential or nerve impulse in the part of the nerve fiber that runs in the organ of Corti and into the spiral lamina. (The main action potential "spike" is generated within the modiolus.) But, even before this, Mr. Goldstein[5] in our laboratory found that there are at least two and probably three components of the "summating potential" that can be revealed successively by progressive asphyxia and various other injurious processes. We are inclined to believe that one of these relatively hardy potentials represents the postulated local excitatory process.

Time does not allow presentation of the evidence and/or the argument by which we showed[2] that, regardless of the potentials we demonstrate on the oscilloscope, summation *must* occur in the excitatory process. Somehow two or even several sound waves (at high frequencies) add their effects to initiate a single nerve impulse. Wever[8] had already arrived at the same conclusion by a somewhat different argument. This much insight we believe we have gained into the nature of the excitatory process.

EXCITATION IN THE COCHLEA

We must picture the cochlea as a whole as a delicate acoustic analyzer that localizes the energy of the sound in different areas according to the frequency of the sound waves. We must also picture it as a delicate electrochemical apparatus, with the energy of metabolic processes at its disposal, that liberates some of this energy in response to mechanical disturbance. The energy initiates a "local excitatory process" that can outlast brief sound waves and perhaps add together the outputs of more than one sensory cell to set up nerve impulses. These nerve impulses run to the brain in a rather complicated set of nerve fibers. Possibly the excitability of the sensory cells is modified by efferent impulses from the brain.

It is small wonder that this complicated and delicate organ is sensitive to injury, be it by too strong mechanical disturbance by very loud sound, or by asphyxia, and to other changes in its chemical surroundings. Perfusion of scala vestibuli with isotonic potassium chloride, for example, reversibly suppresses the cochlear microphonic and the action potentials. Electrical polarization of the basilar membrane increases or decreases the potentials, depending on the direction of the polarization.[7] Even more interesting: we find that any surgical intrusion into the scala media or even loosening the spiral ligament from the bony wall causes a loss locally of both the microphonics and action potentials. Some of these effects are reversible, some are not. We have accumulated many facts that we have not yet assimilated, but from this mass of experimental data we may hope to gain insight, bit by bit, into the various disturbances of excitability within the cochlea that we group together as nerve deafness.

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TRIANGULAR CUPPED LARYNGEAL FORCEPS

LEO SCHWARTZ, M.D.
NEW YORK, N. Y.

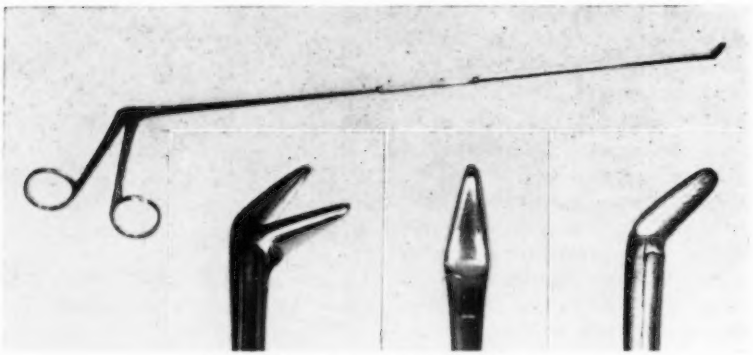

FIG. 1—Triangular cupped laryngeal forceps.

THESE forceps are the culmination of an effort to devise a single instrument that would embrace many of the advantages and eliminate some of the disadvantages of most of the commonly used forceps.

It is hoped that the one presented will, in a large measure, make it unnecessary for the operator to be burdened with a large assortment of forceps in the removal of a majority of the small benign tumors of the vocal cords and in taking biopsy specimens.

The instrument is fashioned so that the biting edges form a *V* with the apex toward the anterior commissure. The bite is linear in contradistinction to the arcuate or circular bite of the round cups and can be used on either right or left cord or both. It is adapted for use in the subglottic area and anterior commissure.

The shaft has been made exceedingly thin or "delicate" and the cups flattened in order to give greater visibility to the instrument when it is placed in the operative area.

Further models of various sizes and various degrees of angulation of the biting end are contemplated.

Mr. Henry Pilling and his associates have been of great assistance in developing the instrument.

I am greatly indebted to Dr. I. M. Schnee of Paterson, New Jersey, for making the presentation for me in my absence from the Academy meeting.

Presented as a *New Instrument* at the Fifty-Sixth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 14-19, 1951, Chicago, Ill.

THE USE OF CORTISONE IN OTOSCLEROSIS

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THE INTRODUCTION of pituitary-adrenocorticotropic hormone (ACTH) and cortisone has shaken pathologic and therapeutic concepts in every field of medical investigation. It is thus inevitable that certain otologic problems will be reinvestigated from the standpoint of these new potent hormonal weapons.

Cortisone, a highly purified extract of adrenal cortex, is one of several compounds that were isolated by Reichstein, Kendall, Hench and their colleagues. The successful therapeutic attack on rheumatoid arthritis by cortisone is now established medical history. Since this initial triumph, innumerable applications of the drug have been made in the treatment of other diseases. The rationale for the therapy still remains a moot question. No adequate theory can explain the therapeutic effectiveness in so many apparently unrelated diseases. In ophthalmology, cortisone has proved valuable in many diseases hitherto considered irreversible and hopeless. The drug has been used with similar success in acute rheumatic fever, in rheumatic pancarditis, and in hitherto fatal skin diseases, including pemphigus and lupus erythematosus disseminatus.

There are several theoretical reasons for the investigational use of cortisone in otosclerosis.

1. The histologic picture of the fixed stapedial-vestibular articulation in the mature otosclerotic state resembles in some aspects the deformation of joint architecture in advanced rheumatoid arthritis. The brilliant results achieved in the treatment of the latter disease and the pathologic analogy between the two would seem to justify the clinical investigation of cortisone in well-defined adult otosclerosis with audiometric and otologic evidence of a fixed stapes.

2. Partial fixation of the stapes which occurs in early cases of otosclerosis may resemble the clinical pathology of acute rheumatoid arthritis. The otosclerotic changes in the annular ligament are not dissimilar to the changes seen in the synovial membranes of some joints. The reversibility, therefore, of the rheumatic process offers a challenge in the potential reversibility of the early otosclerotic process. It brings up the possibility of a fibrolytic activity of cortisone.

3. The salutary effect of cortisone on some of the collagen diseases and on many of the apparently irreversible ophthalmic diseases led to speculation that the drug may have a similar effect on the prevention of progression of the otosclerotic process, not only in the oval window region but in the round window and cochlear ganglion areas.

4. Certain theoretical applications of cortisone in fenestration surgery have been proposed. Fenestral closure is usually due to uninhibited capsular osteogenesis, assuming that all technical

From the Institute for Medical Research, Cedars of Lebanon Hospital, Los Angeles, Dr. Harry Goldblatt, Director, and the Department of Otolaryngology, University of Southern California.

The author should like to acknowledge the assistance of Dr. Seymour J. Brockman. He is also indebted to Drs. Howard P. House and Henry Harris who supplied clinical material and collaborated in the study.

The cortisone (Cortone) used in this study was supplied through the courtesy of Dr. Augustus Gibson, Medical Director, Merck & Co., Inc.

Presented before the Otosclerosis Study Group, Oct. 13, 1951, Chicago, Ill.

details have been dealt with satisfactorily. Physiologic studies of the action of cortisone and ACTH have shown an inhibitory effect on osteogenesis and on the formation of granulations. Consequently this brings up the theoretical possibility of (1) preventing osteogenesis in the fenestration operation and (2) reversal of incomplete closure by fibrolytic activity of cortisone.

The above theoretically encouraging applications of cortisone in otosclerosis should be soberly tempered, however, by the following considerations which might well be considered theoretical objections to the potential therapeutic value of cortisone in otosclerosis.

1. At the present time there is no histologic evidence for a true arthritic pathogenetic picture in the process of otosclerotic stapedial fixation. Essentially this process is not intra-articular but extra-articular. The architectural deformation of the stapedial-vestibular joint is usually due to extra-articular vascular invasion of bone followed by osteoclastic destruction of joint architecture and completed by final osteoblastic new bone formation.

2. There is no clinical correlation between otosclerosis and rheumatoid or other forms of arthritis.

3. The question of fenestral closures has been thoroughly investigated by Lempert and many of his pupils. Indisputable evidence exists to demonstrate that many closures were due (a) to faulty placement of the fenestra in the posteroinferior aspect of the horizontal semicircular canal and (b) to faulty creation of the fenestra from the standpoint of wound margin treatment. Present technics, advocated by Lempert and his associates, have undoubtedly minimized fenestral closures to practically irreducible quanta. Those closures which occur in spite of all technical and anatomical measures, must be considered due to individual biologic tendency to osteogenesis.

In spite of the above objections, on both theoretical and practical grounds, the dramatic demonstration of therapeutic potency of this drug in apparently irreversible diseases elsewhere in the body, makes an investigational approach in otosclerosis a definite necessity.

The following study was approached with no preformed opinions as a very early scanning survey of the potentialities for therapeutic effect in this still baffling disease.

The decision for the employment of cortisone, rather than pituitary-adrenocorticotropic hormone (ACTH) in this particular study was based upon the following facts.

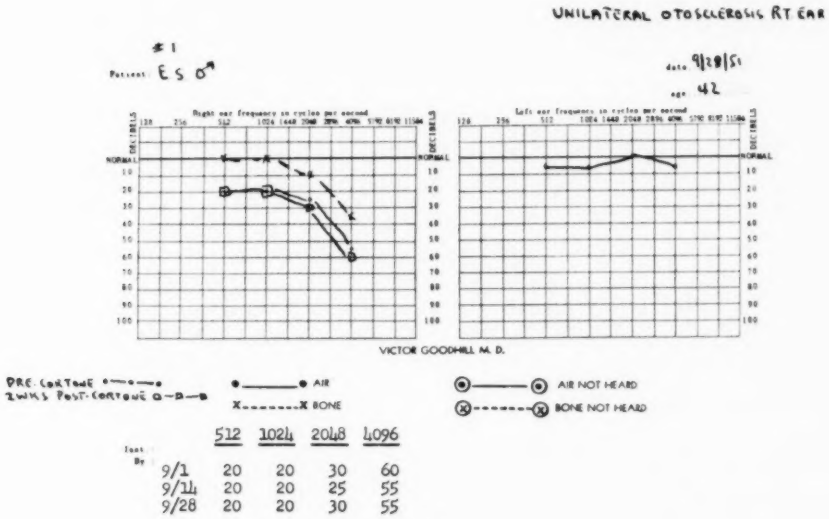
1. The recent availability of *oral* cortisone made such a study a reasonably feasible experimental approach. There are apparently also fewer and milder side effects and sequelae with cortisone than there are with ACTH.

2. The availability of oral cortisone for this investigation was due to the generosity of Merck & Co., Inc. This obviously was a most important reason for the selection of cortisone for this preliminary study of steroids in otosclerosis.

CASE REPORTS

The cases selected for this initial study were drawn from a miscellaneous assortment of otosclerotics. The limitation in available drug prompted the selection of an assorted variety of otosclerotics for the purposes of preliminary study. Obviously insufficient drug was available for the adequate statistical analysis of any one subdivision. The primary purpose was a scanning exploration of the field to enable more intensive and selective group studies later.

The question of dosage was a most difficult one to assess. Great variations in dosage schedules exist in the application of the drug in other diseases. No true therapeutic analogy could be used for a dosage guide, other than the one in use at the present time in rheumatoid arthritis. Inasmuch as most authorities at the present time report obvious therapeutic effects with the administration of but 0.5 gram to 1 gram of the drug with should be used for a beginning yardstick in the otosclerosis study. Further experiences may reveal that the selection of dosage was far too low. The question of side effects, however, was very important also in determining the relatively moderate dosage schedule outlined. In view of the completely unexplored field, it was not deemed advisable to subject patients to great hazards from side effects.



Test Dr	512	1024	2048	4096
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9/14	20	20	25	55
9/28	20	20	30	55

FIGURE 1.

recommendations for dosage schedules not to exceed three or four weeks in any one course, it was decided in this study to use between 1.5 and 2 grams as a rough average in the majority of patients. The duration of administration varied from one to three weeks with an average of approximately ten to twelve days. Inasmuch as this length of time is usually adequate for objective and subjective evidence of therapeutic effect in arthritis and in many of the eye diseases, it was felt that a similar schedule

Case 1. E.S., age 42, white male. Audiometric and otologic evidence of right unilateral otosclerosis. Cortisone, 1,575 mg. administered over a ten-day period. No evidence of audiometric change (fig. 1). No subjective change.

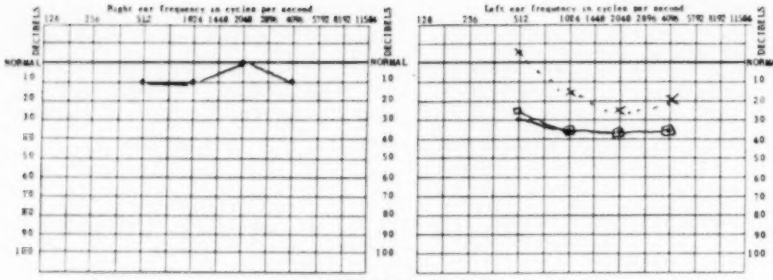
Case 2. O.T., age 51, white female. Otologic and audiometric evidence of otosclerosis, predominant in the left ear (fig. 2). Cortisone 1,600 mg. administered over a ten-day period. No subjective or audiometric change in threshold.

UNILATERAL OTOSCLEROSIS

2

Patient O.T. ♀ date 9/24/51

age 51



● ——— ● AIR
X - - - - - - - X BONE
⊙ ——— ⊙ AIR NOT HEARD
⊗ - - - - - - - ⊗ BONE NOT HEARD

Test By

	512	1024	2048	4096
8/31	30	35	35	35
9/10	25	35	35	35
9/24	25	35	35	35

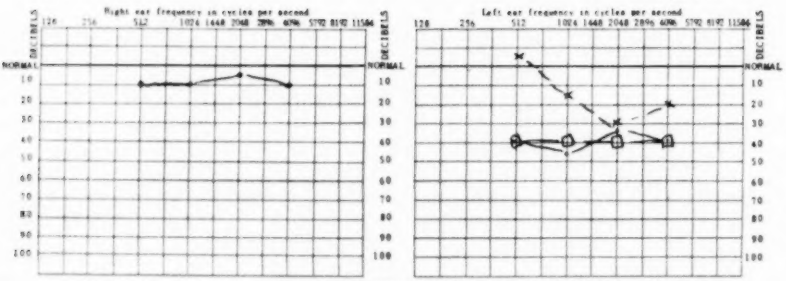
FIGURE 2.

UNILATERAL OTOSCLEROSIS

3

Patient O M J ♀ date 9/21/51

age 26



● ——— ● AIR
X - - - - - - - X BONE
⊙ ——— ⊙ AIR NOT HEARD
⊗ - - - - - - - ⊗ BONE NOT HEARD

Test By

	512	1024	2048	4096
8/31	40	45	35	40
9/7	40	40	40	40
9/21	40	40	40	40

FIGURE 3.

Case 3. O.M.J., age 26, white female. Otologic and audiometric evidence of left unilateral otosclerosis. Cortisone 1,600 mg. administered over a ten-day period. No subjective or audiometric change (fig. 3).

Case 4. R.E.P., age 30, white female. Unilateral right otosclerosis. Cortisone 1,600 mg. over a ten-day period. No subjective or audiometric change (fig. 4).

Case 5. L.M., age 30, white female. Bilateral otosclerosis. Cortisone 1,600 mg. over a ten-day period. No subjective or audiometric change (fig. 5).

Case 6. R.R., age 28, white female. History of bilateral otosclerosis of at least ten years' duration. Right fenestration operation, January, 1951. Excellent audiometric postoperative result. Fenestral closure, April 1951. Cortisone 1,400 mg. administered nine months postoperatively. No audiometric or subjective improvement (fig. 6).

Case 7. E.B., age 27, white female. Otosclerosis, eight years' history, bilateral. Left fenestration operation, 1949. Closure within three months. Revision left fenestration, 1950. Closure of revised fenestra, two months later. May, 1951, cortisone 1,450 mg. administered in a seven-day period. Side effects of dizziness and hot flushes necessitated discontinuance of therapy. No subjective or audiometric change (fig. 7).

Case 8. R.F., age 35, white male. Bilateral otosclerosis. 1,675 mg. cortisone administered over 12-day period. No subjective or audiometric change (fig. 8).

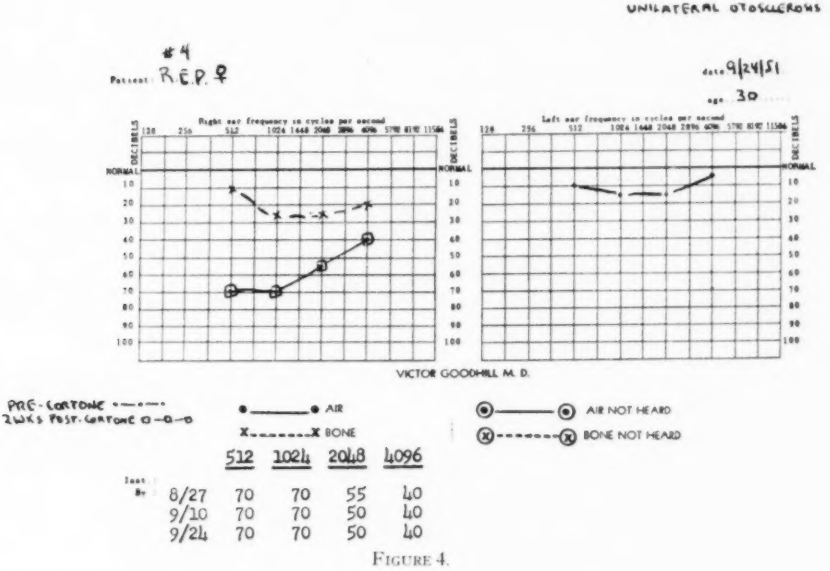


FIGURE 4.

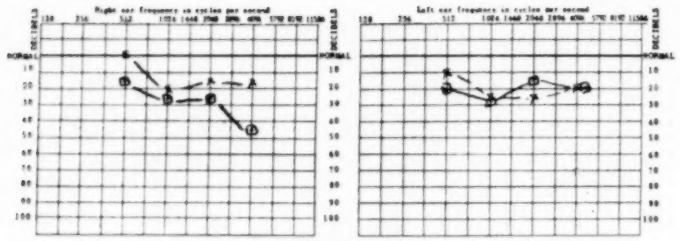
Case 9. H.A., age 30, white male. Bilateral otosclerosis, at least ten years' duration. 1,425 mg. cortisone in ten-day period. No audiometric or subjective change. A second more intensive course of cortisone was given eight months later. This consisted of 300 mg. daily for seven days, a total of 2.1 grams. There was no audiometric or subjective change (fig. 9).

Case 10. E.M., age 36, white female. Bilateral otosclerosis, ten years' duration. Left fenestration, 1944. Fenestral closure six months postoperatively. Revision, 1946. Clo-

BILATERAL OTOSCLEROSIS

#5
Patient L.M ♀

date 9/21/51
age 30



PRE. CORTONE ○—○—○ ●———● AIR
2 WKS POST CORTONE □—□—□ X--------X BONE
⊙———⊙ AIR NOT HEARD
⊗--------⊗ BONE NOT HEARD

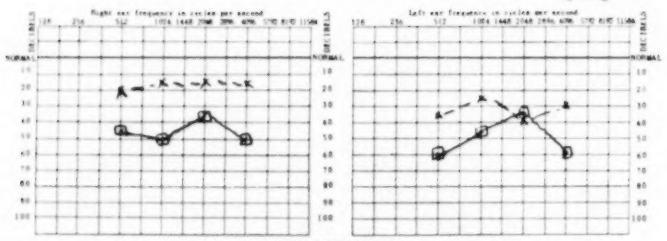
Test By	512	1024	2048	4096	512	1024	2048	4096
8/31	15	25	25	45	20	25	15	20
9/7	20	25	25	40	20	25	15	20
9/21	15	25	25	45	20	25	15	20

FIGURE 5.

POST FENESTRATION RT. 8MO WITH CLOSURE

#6
Patient R R ♀

date 9/7/51
age 28



PRE-CORTONE ○—○—○
2 WKS POST CORTONE □—□—□ ●———● AIR
X--------X BONE
⊙———⊙ AIR NOT HEARD
⊗--------⊗ BONE NOT HEARD

Test By	512	1024	2048	4096	512	1024	2048	4096
8/27	45	50	35	40	60	45	35	60
9/4	45	50	40	40	55	40	35	60
9/17	45	50	35	40	60	45	35	60

FIGURE 6.

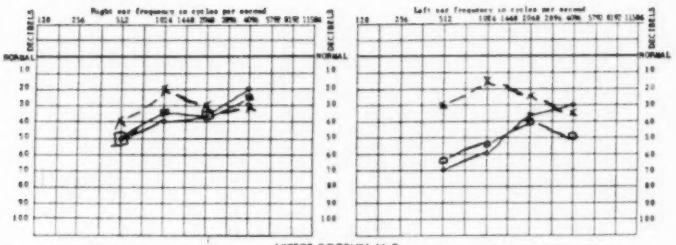
LEFT FENESTRATION REVISED WITH CLOSURE

#7

Patient E.B. ♀

date 9/25/51

age 37



PRE-CORTONE •—•—•

8 MONTHS POST CORTONE o—o—o

•——— • AIR

X-------X BONE

⊙——⊙ AIR NOT HEARD

⊗-------⊗ BONE NOT HEARD

Inst. By	512	1024	2048	4096	512	1024	2048	4096
1/19	50	40	35	20	70	60	35	30
4/3	50	40	35	25	65	55	40	50
9/25	50	35	35	25	65	55	40	50

FIGURE 7.

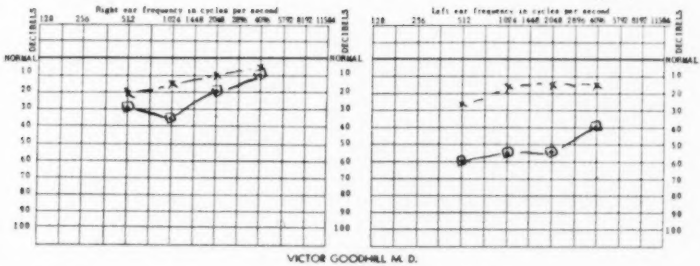
OTOSCLEROSIS

#8

Patient R.F. ♂

date 9/25/51

age 35



PRE-CORTONE •—•—•

2 WKS POST-CORTONE o—o—o

•——— • AIR

X-------X BONE

⊙——⊙ AIR NOT HEARD

⊗-------⊗ BONE NOT HEARD

Inst. By	512	1024	2048	4096	512	1024	2048	4096
9/4	30	30	20	10	60	55	55	40
9/15	30	30	20	10	55	55	55	40
9/25	30	35	20	10	60	55	55	40

FIGURE 8.

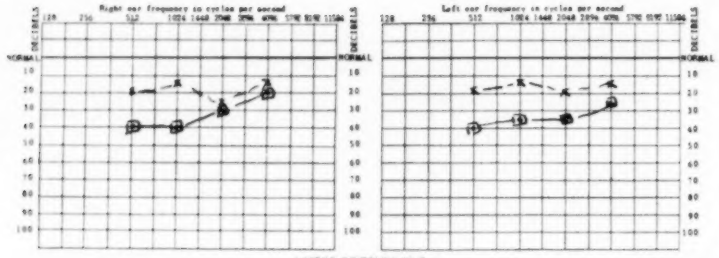
BILATERAL OTOSCLEROSIS

9

Patient H.A. ♂

date 10/5/51

age 30


Right ear frequency in cycles per second
Left ear frequency in cycles per second
128 256 512 1024 1448 2048 2896 4096 5792 8192 11584
DECIBELS
NORMAL
10 20 30 40 50 60 70 80 90 100

VICTOR GOODHILL M. D.

PRE-CORTONE ○—○—○
POST-CORTONE □—□—□

●——● AIR
X- - - - - - - X BONE
⊙——⊙ AIR NOT HEARD
⊗- - - - - - - ⊗ BONE NOT HEARD

Test By	512	1024	2048	4096	512	1024	2048	4096
1/19	40	40	30	20	40	35	35	25
2/9	40	40	30	20	40	35	35	25
10/5	40	40	30	20	40	35	35	25

FIGURE 9.

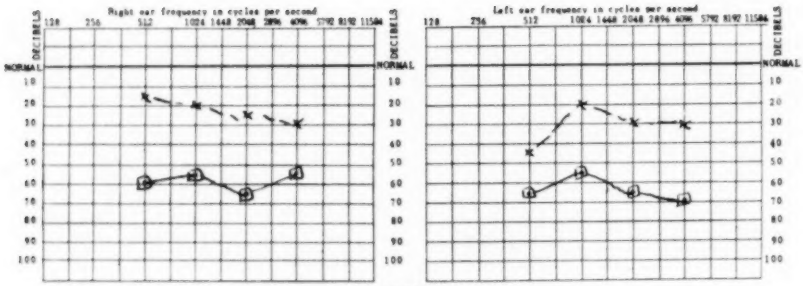
LEFT FENESTRATION REVISED TWO TIMES WITH CLOSURE

10

Patient E.M. ♀

date 9/2/51

age 36


Right ear frequency in cycles per second
Left ear frequency in cycles per second
128 256 512 1024 1448 2048 2896 4096 5792 8192 11584
DECIBELS
NORMAL
10 20 30 40 50 60 70 80 90 100

VICTOR GOODHILL M. D.

●——● AIR
X- - - - - - - X BONE
⊙——⊙ AIR NOT HEARD
⊗- - - - - - - ⊗ BONE NOT HEARD

Test By	512	1024	2048	4096	512	1024	2048	4096
3/30	60	55	65	55	65	55	65	70
4/16	70	60	65	60	65	55	65	70
9/2	60	55	65	55	65	55	65	70

FIGURE 10.

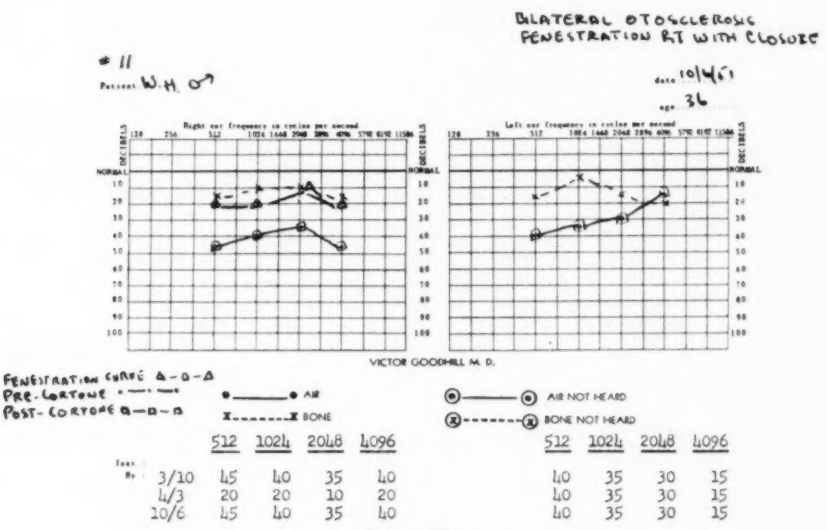
sure four months postoperatively. Third revision, 1948, with closure two months postoperatively. In 1951, 1,400 mg. cortisone over ten-day period. No subjective or audiometric change (fig. 10).

Case 11. W.H., age 36, white male. Bilateral otosclerosis, four years' duration. Right fenestration, March 6, 1951. Cortisone started March 10, 1951, 2,300 mg. administered during the next three weeks. During the course of this administration acute appendicitis developed. April 3, 1951, audiometric studies showed an excellent postoperative level. June 25, 1951, there was a 25 db. level in the speech frequencies. In August there was a negative fistula test and a drop in the speech frequencies to 40 db.

We have here a patient who received cortisone for approximately three weeks, immediately postoperatively. In spite of an originally excellent result, fenestral osteogenesis occurred at the usual rate, attaining complete closure in four months. This was definite evidence that immediate postoperative administration of cortisone would not inhibit fenestral osteogenesis.

A second course of cortisone was given intensively in September, 1951. This consisted of 300 mg. daily for seven days, a total of 2.1 grams. There was no audiometric or subjective change (fig. 11).

Case 12. A.S., age 27, white female. Bilateral otosclerosis, worse in the right ear. Strong familial history. 1,000 mg. cortisone given in a one-week period. No subjective or audiometric improvement (fig. 12). This was followed one month later by a right fenestration operation with an excellent immediate result.



Test By	512	1024	2048	4096	512	1024	2048	4096
3/10	45	40	35	40	40	35	30	15
4/3	20	20	10	20	40	35	30	15
10/6	45	40	35	40	40	35	30	15

FIGURE 11.

Case 13. J.F., age 26, white male. Bilateral otosclerosis. 1,575 mg. cortisone in a ten-day period. No subjective or audiometric change (fig. 13).

A second course of intensive cortisone was administered four months later. This consisted of 300 mg. daily for seven days, a total of 2.1 grams. There was no audiometric or subjective change.

Case 14. R.S., age 47, white male. Bilateral otosclerosis. Left fenestration, 1950. Fenestral closure two months later. Left fenestration revision, 1951, with no hearing gain. Cortisone 1,575 mg. over 12-day period. No subjective or audiometric change (fig. 14).

A second course of intensive cortisone was administered two months later. This course, 300 mg. daily for seven days for a total of

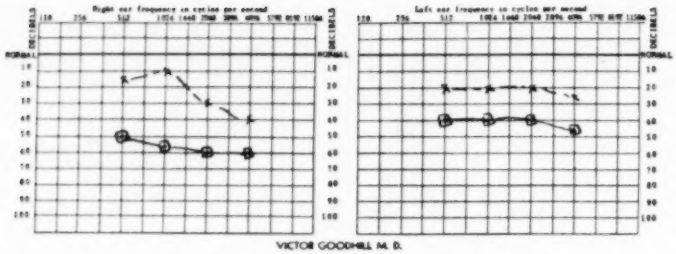
BILATERAL OTOSCLEROSIS WORSE ON THE RIGHT

12

Patient A. S. ♀

date 4/14/51

age 27



PRE-CORTONE •——•——• ● ——— ● AIR ⊙ ——— ⊙ AIR NOT HEARD

2 WKS POST-CORTONE □—□—□ X - - - - - - - X BONE ⊗ - - - - - - - ⊗ BONE NOT HEARD

Test	512	1024	2048	4096	512	1024	2048	4096
Dr. 3/28	50	55	60	60	30	30	30	30
4/11	50	60	60	60	30	25	30	30
4/14	50	55	60	60	30	30	30	30

FIGURE 12.

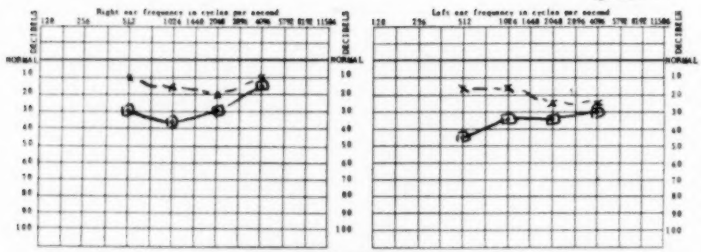
BILATERAL OTOSCLEROSIS

13

Patient J. F. ♂

date 10/5/51

age 26

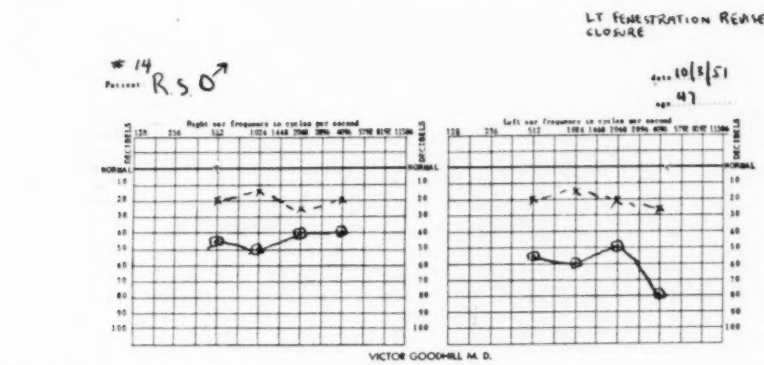


PRE-CORTONE •——•——• ● ——— ● AIR ⊙ ——— ⊙ AIR NOT HEARD

POST-CORTONE □—□—□ X - - - - - - - X BONE ⊗ - - - - - - - ⊗ BONE NOT HEARD

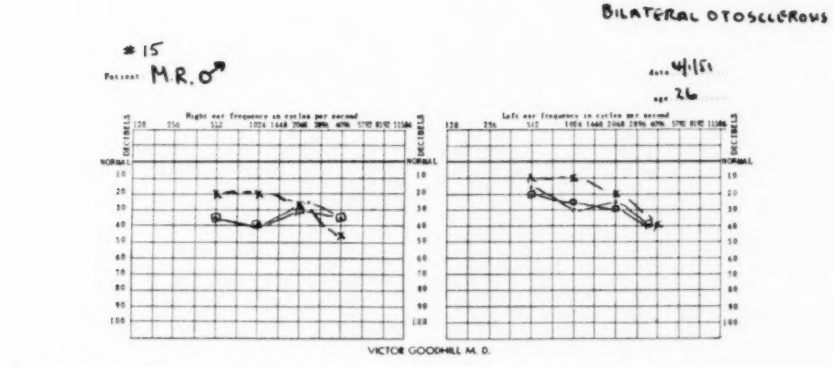
Test	512	1024	2048	4096	512	1024	2048	4096
Dr. 8/1	30	35	30	15	45	35	35	30
9/10	30	35	30	15	45	35	35	30
10/5	30	35	30	15	45	35	35	30

FIGURE 13.



Test By	512	1024	2048	4096	512	1024	2048	4096
8/31	45	50	40	40	55	60	50	80
9/8	45	50	40	40	55	60	50	80
10/4	45	50	40	40	55	60	50	80

FIGURE 14.



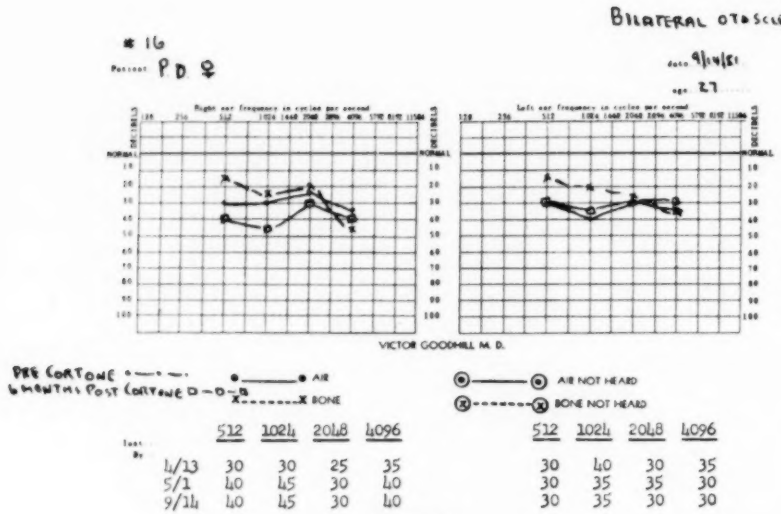
Test By	512	1024	2048	4096	512	1024	2048	4096
1/22	35	40	25	35	15	25	30	40
2/15	35	40	30	35	20	25	30	40
4/1	35	40	30	35	20	25	30	40

FIGURE 15.

2.1 grams, produced no audiometric or subjective improvement.

Case 15. M.R., age 26, white male. Bilateral otosclerosis. Cortisone 1,775 mg. over a 12-day period. No subjective or audiometric change (fig. 15).

Case 16. P.D., age 27, white female. Otosclerosis, bilateral. Cortisone 1,625 mg. over 15-day period. No subjective or audiometric change (fig. 16).



Test By	512	1024	2048	4096	512	1024	2048	4096
4/13	30	30	25	35	30	40	30	35
5/1	40	45	30	40	30	35	35	30
9/14	40	45	30	40	30	35	30	30

FIGURE 16.

SIDE EFFECTS

No serious effects were encountered. Many of the patients experienced temporary euphoria with occasional temporary subjective impressions of increased hearing acuity. These subjective impressions were all transitory and none of them were confirmed by audiometric examinations. A few patients had moderate gains in weight which were quickly controlled by sodium depletion and the use of mild diuretics. There were no other untoward problems in the administration of the drug.

SUMMARY

1. In view of the theoretical similarity between stapedial fixation in otosclerosis and joint fixation in arthritis, cortisone was given a preliminary experimental trial.

2. A group of miscellaneous otosclerotics was treated with oral cortisone. This included unilateral, early bilateral, classical bilateral, postfenestration closure, postrevision closure, and immediate postfenestration cases.

3. Experimental and clinical evidence of inhibition to fibrosis and osteogenesis prompted the trial of the drug in one case of otosclerosis immediately postoperatively.

4. In none of the sixteen cases of otosclerosis did cortisone produce any changes in hearing threshold. Temporary subjective improvements due to elation and euphoria were not confirmed audiometrically and did not persist.

5. Further studies with larger doses and with ACTH are contemplated.

CONCLUSIONS

1. There is theoretical evidence to justify the clinical investigation of the effect of cortisone on otosclerosis.

2. Preliminary studies, however, of sixteen patients showed no evidence of hearing improvement in any of the patients.

3. Further studies with cortisone, ACTH, and other steroid drugs should be encouraged, but caution is indicated both in investigative technics and in interpretation of findings.

TRANSACTIONS
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All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

Editorials

ATLASES OF PATHOLOGY

THE fourth edition of the Atlas of Otolaryngic Pathology and a revised and expanded Atlas and Text on Ophthalmic Pathology, published under the joint sponsorship of the Academy and the Armed Forces Institute of Pathology, are now in the hands of the publishers and will probably be ready for distribution by the time of the annual meeting of the Academy in October.

The Atlas of Otolaryngic Pathology is practically new. Many conditions not shown in previous editions have been included in the present volume, and new photomicrographs selected and prepared by Dr. Muriel Raum in the Institute and edited by Col. J. E. Ash are substituted for cuts that made up previous Atlases. Much new material on the pathology of the ear was contributed by Dr. Edmund P. Fowler, Jr., of New York from his laboratory in Columbia University, and the great resources of the Tumor Registry on Otolaryngic Pathology yielded a wealth of excellent teaching material that had not previously been fully exploited. The Atlas of Otolaryngic Pathology will be published under the sponsorship of the National Research Council.

The Textbook-Atlas of Ophthalmic Pathology contains all new photomicrographs of ocular pathology selected from the thousands of accessions now in the Registry and prepared by Helenor Campbell Wilder, who also participated in the preparation of the first, second and third editions of the Atlas. The addition of the text was suggested by Dr. Jonas Friedenwald, who contributed several chapters and served as chief of the editorial committee. This work is unique in publications of its kind and is designed to aid the student and the pathologist in understanding the intricacies of disease processes peculiar to the organ of vision.

The production of the Atlases was undertaken by the staff of the Army Medical Museum at the request of the Academy and the first editions appeared in 1938. Since the establishment of working relations between the Academy and the Army Medical Museum in 1921, an uninterrupted flow of pathologic material from civilian surgeons has passed through the diagnostic facilities of the Museum and has been added to the registries of pathology until now the Armed Forces Institute of Pathology, as it is currently called, has the most compre-

hensive collection of pathologic specimens of eye, ear, nose and throat in the country.

When the registries were established there were few eye pathologic laboratories in the country. General pathologic laboratories were not staffed to do eye pathology, and the staff at the Army Medical Museum, aware of this situation, established a diagnostic service for eye pathology with Dr. Frederick Verhoeff of Boston and Dr. Jonas Friedenwald of Baltimore as consultants. General laboratories were, however, able to serve otolaryngology's needs, as the type of pathologic material submitted was, as a rule, not different from general pathologic specimens submitted for diagnosis. As the need for diagnostic service in otolaryngologic circles was less urgent, the facilities of the Museum were not utilized for this purpose until several years after the eye service was inaugurated.

The present situation is far different from that of thirty years ago. The study of histopathology has become one of the major subjects in otolaryngology and in ophthalmology. The importance of this subject in the medical curriculum has steadily increased and facilities for study and research and for teaching histopathology in schools and in hospitals have been developed in every medical center. The Academy was the first of the clinical societies to foster the development of specialty tumor registries and to provide through them slides for study by graduate students. The facilities of the Army Medical Museum were made available through the courtesy of Brigadier General Elbert de Coursey, then Curator of the Museum, and now again today in charge of the laboratory.

This project alone is sufficient to identify the Academy as an institution devoted to education and instruction. Other activities have since been added to the Academy's educational program that further prove its standing in the foremost ranks of extracurricular educational institutions. The wisdom and foresight of the Academy Council in providing more facilities for teaching histopathology is proved by the popularity of the courses given annually in the Instruction Courses and by the interest shown in the presentation of rare cases at the opening of each scientific session on ophthalmology.

The Academy now well recognizes the excellence of histopathologic laboratories scattered over the country, and in association with the Armed Forces Institute of Pathology it will sponsor a coalition of resources and a collaboration of efforts to provide a comprehensive pathologic service to civilian surgeons as well as to meet the needs of the Armed Forces and the Veterans Administration.

The publication of the Atlases will mark the fulfillment of a unique project in medical education. At the annual meeting of the Academy in 1952 this important advance in medical teaching will be appropriately celebrated.

W. L. BENEDICT, M.D.

XVII INTERNATIONAL IN 1954

The XVII International Congress of Ophthalmology will take place at the Waldorf-Astoria Hotel in New York City the week beginning September 12, 1954 marking 97 years after the meeting of the First International Congress held in Brussels in 1857. This will be the second time in 78 years that the Congress has been held in the United States, the previous meeting being the V Congress held in New York in 1876. The last meeting was held in London in 1950.

The meetings of the International Congress take place every four years except for interruptions caused by wars. They are conducted under the authority

of the International Federation of Ophthalmological Societies, one of the oldest of all international organizations. The broad aim of the Federation, which is composed of delegates sent by ophthalmological societies of many countries, is to promote the science of ophthalmology throughout the world.

Dr. Bernard Samuels of New York will serve as president of the XVII International Congress with Dr. John H. Dunnington, also of New York, as vice-president, and Dr. William L. Benedict of Rochester, Minnesota, as secretary general.

The invitation to the Congress to meet in this country was the joint action of four national groups: the American Ophthalmological Society, the Section on Ophthalmology of the American Medical Association, the Association for Research in Ophthalmology, and the American Academy of Ophthalmology and Otolaryngology.

It is tentatively planned for the annual meeting of the American Academy of Ophthalmology and Otolaryngology to take place at the Waldorf-Astoria Hotel the week following the XVII International Congress.

The International Council has met recently in Oxford and has selected the subjects which will be discussed at the Congress, namely, *Primary Glaucoma* and *Etiology of Uveitis*. On each of these subjects there will be three main papers.

On *Primary Glaucoma* these will be:

1. Etiology and General Considerations. Dr. Derrick Vail, Chicago, Illinois
2. Medical Therapy. Dr. R. Thiel, Frankfurt a/Main, Germany
3. Surgical Treatment. Dr. Gabriel Sourdille, Nantes, France

On *Etiology of Uveitis:*

1. General Considerations. Dr. Alan C. Woods, Baltimore, Maryland
2. Allergic Factors. Dr. Norman Ashton, London, England
3. Virus Factors. Dr. Vittoriano Cavara, Rome, Italy

Those who wish to present dissertations or to participate in discussions should communicate with the secretary general. English, French and Spanish are the official languages of the Congress but any language may be used. Communications should not be longer than 2500 words and will be read in abridged form. On submission each paper should be accompanied by a resume of about 250 words in each of the three official languages. Papers published previous to the meeting of the Congress are not acceptable. The Publication Committee reserves the right of rejection.

Scientific and clinical demonstrations will be scheduled and there will be scientific and industrial exhibits.

It is hoped that this meeting will be as stimulating as previous Congresses. Delegates from more than fifty countries are expected. This will give a rare opportunity for making new acquaintances and renewing old ones.

Further information will be published from time to time. All correspondence should be directed to Dr. William L. Benedict, Secretary General, 100 First Avenue Building, Rochester, Minnesota.

MANUALS

The publication and distribution of Manuals have become an important aspect of the educational program of the Academy. The continuing demand for them is convincing evidence that they fill a definite need.

Most of the Manuals were originally planned primarily for use in connection with the Home Study Courses, but their distribution is by no means limited to physicians registered for those courses. They are widely used by medical schools, hospitals and the Veterans Administration as aids in postgraduate medical training, and by clinicians in our specialties.

From the beginning the concept of the Manuals has been that they should contain material assembled and condensed from textbooks and current literature, with brief presentations of some of the newer ideas and theories that may not have been generally accepted, and may or may not have been published in the journals. It is planned to keep them up to date by revisions every two or three years. In this way frequent additions and deletions will keep the publications abreast of current thinking and best adapted to supplement other available reference material.

Eleven Manuals and Monographs on subjects related to ophthalmology and six on otolaryngology are in current use. Others are in preparation on:

Glaucoma

Examination of the Labyrinth in Relation to Its Physiology and to Its Nonsuppurative Diseases.

As the need becomes apparent an effort will be made to publish Manuals on additional subjects.

LETTER TO THE SECRETARY

July 21, 1952

Dear Dr. Benedict:

On behalf of the Ophthalmological Society of Australia, I have the greatest pleasure in accepting the kind offer of the Academy of a copy of your coloured motion picture, "Embryology of the Eye."

Having read the review of this film in the American Journal of Ophthalmology for 1950, we know in advance the excellent qualities of the film, and we are looking forward to seeing it and sending it around to meetings of our various State branches.

To afford Dr. Algernon B. Reese an opportunity of making a formal presentation of the film we are arranging an official screening at 4 p.m. on Monday, August 25. There will be present members of the medical faculty of the Melbourne University and, I am very happy to say, Professor Ida Mann, late of Oxford, who will comment on the film technically. Prof. Mann lives in Perth, W.A., and she is coming to Melbourne two days earlier than she intended especially for this purpose.

Again, may I repeat, we appreciate most sincerely this splendid gift from the Academy.

With best wishes,

Yours sincerely,
G. A. Brew, M.D.
President
Ophthalmological Society
of Australia

IMPORTED TONOMETERS

With the re-establishment of normal trade relationships between Europe and the U.S.A., new Schiötz tonometers of foreign manufacture have appeared on the American market. A good many of these instruments, at first glance, show signs of improvement in design and workmanship over the older European models. On close inspection about 25 per cent of the new instruments are found to correspond fairly well to the Schiötz standard; the majority, unfortunately, differ in design so much from the standard or are so poorly constructed as to make one despair of the production of Schiötz tonometers ever becoming a standardized procedure.

Inaccuracy in the construction of the base of the plunger is the most common violation of Schiötz' specifications among the recently imported instruments. These specifications are clearly stated in Schiötz' first article on tonometry (Arch. f. Augenh. 52:401, 1905): "It (the plunger) has a diameter of 3 mm. The base which is intended to rest on the cornea, is concave with a radius of 15 mm." After extensive trials of different plungers on human eyes the Committee on Standardization of Tonometers discovered that one other dimension of the plunger exerted a significant influence on the tonometric reading, the shape of the edge formed by the meeting of the concave base with the cylindrical wall. Schiötz had not specified the shape of this edge in writing, but by measuring the optical profile of a number of original, well-preserved Schiötz plungers the Committee found it to be rounded off to a radius of curvature from 0.212 to 0.300 mm. Adding this measurement to Schiötz' description completely defined the performance of the plunger on the human eye. That, in principle, is the procedure by means of which the Committee has arrived at specifications for all the critical properties of the Schiötz instrument. The certification procedure as performed by Electrical Testing Laboratories of New York, consists of physical measurements of all the critical properties of new tonometers.

Among the imported tonometers one encounters combinations of widely divergent plunger thicknesses (2.90 to 3.15 mm.), base curvatures (from flat to strongly concave) and base edges (0.04 to 0.40 mm. radius or even conical shape). Since the base of the plunger establishes the first contact with the examinee's eye and causes the indentation which is the basis of tonometry, variations in the dimensions of the base have a profound influence on the tonometric reading. A difference in plunger thickness of 0.05 mm. (that is, the difference between a plunger of 3.00 and one of 2.95 mm. diameter) causes a difference in the tonometric reading of about 0.62 scale units. The readings obtained with a blunt-edged plunger (0.40 mm. radius) are about 2 scale units higher than the readings obtained with a sharp-edged plunger of otherwise identical dimensions. These multiple, partly synergistic and partly antagonistic variations of the plunger base in some of the recently imported tonometers have posed serious problems for the American checking stations.

A special case obtains in tonometers of German manufacture covered by a "Prüfungschein," that is a certificate issued by the University of Tübingen. This Committee has been asked repeatedly to give an opinion on the relative values of the German and the American certification procedures.

The German certification procedure is the outgrowth of one of the earliest

TABLE I

Identification number		2047 3035 standard	373/3071
Weight of plunger assembly, gm., at scale reading 5		5.47	6.16
Shape of base curvature of edge, mm.		concave 0.212	flat 0.168
Reading on microgauge, scale units	0	0	1.0
	3	2.9	4.1
	6	5.9	7.2
	9	8.9	10.2
	12	11.9	13.2
	15	14.9	16.2

attempts at standardization of tonometers, the work of Arnold and Karpow of the University of Tübingen (Klin. Monatsbl. f. Augenh., 71:603, 1923), who had observed that the readings obtained with three Schiötz tonometers of Norwegian (Jacobson) manufacture differed as much as 3 scale readings on the same human eye. Variations in the physical characteristics of the three instruments were recognized to be the cause of the difference in their practical performance. These variations were accepted as unalterable facts and a laboratory method was sought that would indicate the combined effect of these variations upon the tonometric reading. After trying various mechanical devices as well as pigs' eyes, the authors finally arrived at the following standardization procedure: A rubber membrane (thickness and other qualities not stated) was tied over the end of a 15 mm. pipe which was connected with a water manometer. The pressure in the water manometer was adjusted so that an original Schiötz tonometer calibrated by Schiötz himself gave a reading of 5 scale units with the 7.5 gm. load when placed centrally on the rubber membrane. The standard tonometer was then removed and replaced with the unknown tonometer and the reading noted. Any deviation from the value 5 was recorded as a fault, again expressed in scale units. In more recent years the test was extended to include readings with the 5.5 and the 10 gm. weights.

The validity of this procedure may be tested in a number of ways. Since the clinician is primarily interested in the performance of a given instrument on a representative group of human eyes and particularly on human eyes whose tension is not materially altered by repeated, brief, applications of the tonometer, the method of direct comparison of instruments calibrated by the Tübingen procedure and a certified tonometer of American manufacture was chosen. An example of such a comparison is shown in the accompanying table, 373/3071 being the imported, Tübingen-certificated instrument and 2047/3035 being the standard (table I). The tonometric study was made on glaucomatous patients, thoroughly familiar with the procedure of tonometry, by a resident physician experienced in tonometry but not familiar with the purpose of the test. Both instruments were used with the 7.5 gm. load, one just a few seconds after the other. The sequence of application was reversed from standard first and unknown second to unknown first and standard second every time five eyes had been tested. The results expressed in scale units are shown in figure 1. A considerable scatter of the results is noted which is largely due to the inaccuracies of the process of tonometry aside from the difference in the physical characteristics of the two instruments. A straight line can be fitted to the data without much difficulty. This straight line indicates a rather consistent difference between the two instruments of a magnitude of from 2.5 to 3 scale units. The certificate accompanying the German instrument indicated a fault of 1 scale unit.

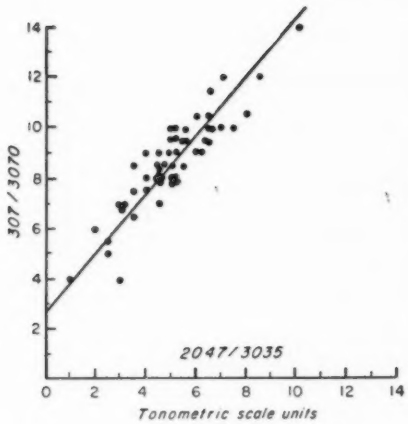

FIGURE 1.

Similar gross discrepancies have been encountered between other Tübingen-certificated instruments and the standard. When such gross discrepancies occurred, the physical characteristics of the plunger of the German instrument differed markedly from those of the standard. If, on the other hand, the German instrument in its physical characteristics corresponded fairly well to the standard, the direct comparison on human eyes usually bore out the result of the German calibration procedure. These observations suggested some similarity but no close parallelism between the indentation of the human eye and that of the rubber membrane made by the tonometer. The matter was investigated further by comparing the performance on human eyes, as well as on rubber membranes, of two tonometers which differed only in one respect, such as plunger weight or plunger thickness. A small study of this kind showed the human eyes to be more sensitive in bringing out a difference between the two tonometers than the rubber membrane. A difference in plunger assembly weight, for example, produced a greater and more consistent difference between the averages of the readings obtained with the two instruments on human eyes than on the rubber membrane. The behavior of the rubber membrane can thus at best be described as qualitatively similar to that of the human eye.

It appears from the foregoing that tests on rubber membranes are of very limited value in the standardization of tonometers. Because of the low test-retest consistency of readings taken on the same rubber membrane with the same tonometer some investigators have rejected this method altogether. Others attach some value to rubber membrane readings in estimating the combined, additive or subtractive, effect of multiple coexistent deviations. If these deviations are fairly well balanced as to direction (increasing or decreasing the depth of the indentation) and if each deviation is small in magnitude, the result of the rubber membrane test may be expected to come fairly close to the result obtained on human eyes.

In conclusion, uniformity of performance on the human eye is more certain to result from painstaking uniformity in tonometer construction than from the selection, from physically discrepant tonometers, of those instruments whose discrepancies, on the basis of rubber membrane tests, seems to balance each other.

COMMITTEE ON STANDARDIZATION OF TONOMETERS

In Memoriam

Paul Mitten Allis.....Lewiston, Pennsylvania
August 10, 1952

Malcolm Percival Andrews.....Manitowoc, Wisconsin
July 29, 1952

Ralph Edwin Boice.....Toledo, Ohio
March 30, 1952

Morgan Cryder Davies.....Columbus, Ohio
May 26, 1952

Thomas W. Davis.....Winston-Salem, North Carolina
June 3, 1952

Samuel Davidson Evans.....Pittsburgh, Pennsylvania
June 1, 1952

Talbot John Gorrell.....Chicago Heights, Illinois
April 14, 1952

Herman Paul Grossman.....Providence, Rhode Island
June 5, 1952

Frederick Smith Halsted.....Denver, Colorado
May 1, 1952

William Aloysius Kennedy.....St. Paul, Minnesota
April 30, 1952

Hal Ellsworth Marshall.....Wichita, Kansas
June 15, 1952

Everett Crockett Moulton.....Ft. Smith, Arkansas
January 19, 1952

William Sassaman Sutherland..Philadelphia, Pennsylvania
March 14, 1952

Louisa Paine Tingley.....Boston, Massachusetts
July 16, 1952

Vivian Parley White.....Salt Lake City, Utah
May 13, 1952

INDUSTRIAL OPHTHALMOLOGY

INTRODUCTORY STATEMENT

One of the most beautifully organized medical departments in the country is that of the Oak Ridge Laboratory. It has been the privilege of the secretary of the Joint Committee to be in close touch with this interesting development, and right from the beginning, an intense interest in eye problems has been shown by Dr. Jean Felton, both in the program of pre-employment, visual testing and refracting for the job and in the extra care taken in cases involving pathologic conditions. It is a privilege, therefore, to be able to present this paper with its excellent illustrations.

Various members of the Academy have requested that the paper prepared by the Secretary for the Pan-American Congress of Ophthalmology in Mexico City be presented in this section.

HEDWIG S. KUHN, M.D.
Secretary, Joint Committee
Industrial Ophthalmology

VISUAL HEALTH MAINTENANCE AT AN ATOMIC ENERGY LABORATORY

JEAN SPENCER FELTON, M.D.
OAK RIDGE, TENN.

IN the decade nearing completion, the American people—and the world—have seen the Atomic Age grow from its modest speculative beginnings to its current portentous adolescence. Mindful more of the awesome wonders of nuclear energy, the followers of the newscasts and columns have not been aware of the quiet entry of this new endeavor into the nation's family of gigantic industries.

Simulating some industrial procedures, creating others anew, and altering many traditional practices, atomic energy at production levels has made physiologic demands on its workers requiring unequalled safety and health maintenance measures. The manufacturing plants and research laboratories have established broad industrial medical services to render a good preventive medical or constructive health program. That it is safer to work in such an installation than to spend the time at home testifies to the care given the employees by the physician, the health physicist, and the safety engineer.

The Research Laboratory

A segment of such a program involves the eyes of the worker. The locale of this particular health effort to be described is the Oak Ridge National Laboratory, an installation of over 3,000 employees. Engaged in vital defense work and in basic or fundamental research in nuclear energy, the scientists, technicians, craftsmen, and service personnel of this laboratory are executing cooperatively a mission that calls for the development of many new reactors, the solution of flight by nuclear energy propulsion, the continuing production of radioisotopes, and the search for new truths in the several disciplines of the physical and biologic sciences. Placed on jobs of wide visual demands, this group that ranges from theoretic physicists to custodians, requires a planned scheme of sight conservation, necessary for the preservation of difficult-to-acquire research and supportive talents.

The Early Program In Vision Conservation

During the frenetic, security-bound war years in Oak Ridge, the time-frayed Snellen Chart was used for the testing of employment candidates, an infre-

Medical Director, Oak Ridge National Laboratory. The work described was performed under the auspices of the Atomic Energy Commission.

quent foreign body was removed from an eye, and industrial nurses "passed out" safety glasses on request. From this effort without direction or individualization has grown a modern multiphasic approach to eye protection.

Testing Program

The procurement in 1946 by the Health Division shortly after war's end of a stereoscopic vision testing device marked the first step toward modernization in this health segment. The apparatus was placed in a specially built-in portion of the counter of the Reception-Record Room (fig. 1). With the acquisition of this device, occupational visual analyses were indicated, and from early work sheets, visual performance profiles were developed (fig. 2) which demonstrate the standards required by certain jobs.

In a research laboratory the work is highly diversified, and each position must be analyzed individually to determine the maximum requirements for visual correction and protection. If, as an example, one considers laboratory technicians alone, of which there are 400, one finds work assignments ranging from glass-blowing to observing molten metals; from testing tensile strength of alloys in hydraulic machines to qualitative analysis of irradiated samples of chemical compounds; from reading and recording data from control instruments to processing film used in radiation badge meters; and from preparing intricate chemical equipment to conducting spectrophotometric analyses. A special knowledge of the visual needs and the eye hazards of these widely differing jobs is required.

FIG. 1—Location of stereoscopic vision testing instrument in Record Section of Health Division.

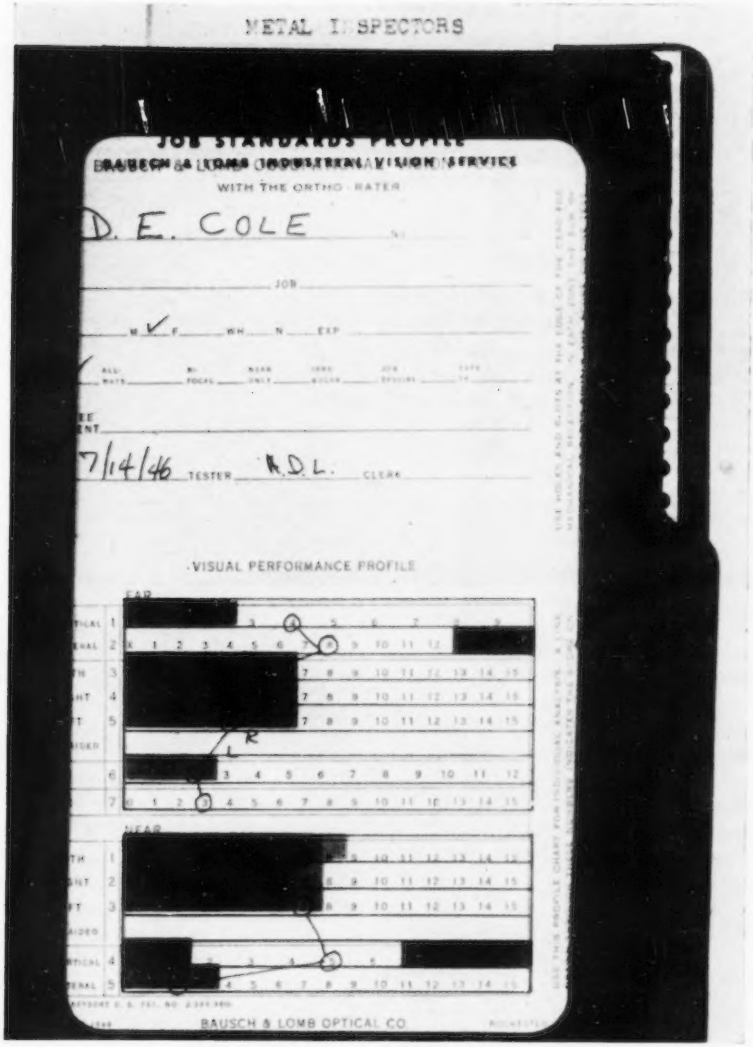

FIG. 2.—Visual performance profile for metal inspectors.

Currently, the visual test battery is being used in several situations. First, all prospective employees are examined. If the visual skills are limited and the individual must be restricted on his job, a notation is made on his physical examination report form to the effect that he *is not to be placed in work requiring acute vision.** Secondly, this type of examination is repeated at the time of periodic physical examinations, as part of special examinations conducted on visually limited persons, physical appraisals for retirement, or retention, at the age of 65 years, special annual examinations on key personnel, or at the termination of absence due to illness if the cause of the absence involves the eyes in any way. Third, it is the standard practice of the Health Division to test the vision of each employee reporting for care of an occupationally incurred injury to an eye. Evidence of persisting partial loss of vision obviously is pursued further. Fourth, when application is made for an Atomic Energy Commission Motor Vehicle Operator's Permit, the vision is tested and the candidate approved or disapproved for the permit in accordance with his meeting the visual requirements for driving.

56 FIELD CLINICAL LAB PROCEDURE		
56 SPECTACLES ISSUED PLANO		35
57 SPECTACLES ISSUED PRES FIRST VISIT		32
58 SPECTACLES ISSUED PRES REVISIT		33
59 ORTHORATER EXAMINATION		176
60 FIELD ORTHORATER EXAMINATION		19
61 PARTIAL REPLACEMENT		13
62 ADJUSTMENT OR REPAIR		24
63 ADVISORY SERVICE		33
OCCUPATIONAL VISION SECTION PROCED	365	

FIG. 3—Summation of clinical procedures as noted on monthly machine records report.

Injuries of the Eye

The occupational file of a research laboratory crosses the full spectrum of job families, so that different types of eye injuries will be encountered. Chemical burns of the eye will be encountered, caused by splash contact with any one of the myriad of raw materials, intermediates, or end products. Because of the great number of maintenance personnel, actinic conjunctivitis will be seen not in the welders but, as always, in the bystander who unwisely watched a welding process, and foreign bodies will be found among those working outdoors. Infrequently through failure to use occupational eyewear, broken pieces of spectacle lenses will be removed from the eye. In all chemical injuries, after initial staining with fluorescein solution and inspection, the eyes are irrigated with sterile saline, maintained in a three gallon carboy in a special cabinet approximately six feet from the floor. If the eye has been splashed by liquids con-

* This method of job and capacity matching has permitted the employment of many visually impaired persons whose work performance has been excellent.

taining radioactive materials, the health physicist is called in to survey the extent of the contamination before and after cleaning procedures take place.

Statistical records are maintained by means of mark-sense punch cards, and end-of-the-month summations are received from the Machine Records Section (fig. 3).

Occupational Eyewear Program

Of greatest importance in any industrial health service is the preventive activity which aims at the identification of and protection against health hazards. A small area with a complete office and waiting room are maintained[1] for the execution of the following functions:

1. Determination of eye protection needs.
2. Verification of prescription as to job visual requirements.
3. Taking of measurements for frame size.
4. Verification of authorization for issue.
5. Preparation of order form to optical manufacturer.
6. Completion of record form of prescription and frame specifications.
7. Verification of lenses and frames on receipt from manufacturer.
8. Notification of employee of receipt of glasses.
9. Fitting of eyewear.
10. Rendering of instructions regarding maintenance of spectacles.
11. Preparation of cost accounting form.
12. Completion of entries in Clinical Record of service rendered.
13. Maintenance of stock items and parts for issue of "plano" glasses.
14. Replacement of parts and adjustment of safety spectacles after issue.
15. Maintenance of liaison with local ophthalmologists.
16. Interpretation to employee of his visual requirements.
17. Courtesy adjustment service of personal eyewear.

Special instruments are needed for this type of service and include the heating cone, stamping machine for gold leaf identification imprint on frames and case, and vertometer.

Because of the variety of jobs present in the Laboratory, the needs for several types of protective eyewear are multiple, and at the present time the Ophthalmic Dispenser has available 11 different types of personal protective eye devices (fig. 4) for use by Laboratory personnel. These are as follows:

1. One piece tinted plastic eye shield —available in various shades.
2. Semi-cup clear plastic spectacle goggle.
3. One piece clear plastic eye shield.
4. Cobalt melter's glasses.
5. Polaroid goggles.
6. Filter mask with prescription ground lens inserts.
7. One piece tinted plastic eye shield with side protection.
8. Welder's goggles.
9. Safety spectacles—cellulose acetate frame.
10. Safety spectacles—cellulose acetate frame, tinted.
11. Safety spectacles—didymium lens.

Corrective lenses are often needed for wear in the filter masks, so special masks were made that would allow the mounting of lenses behind the facepiece.

Some of the Laboratory welders required corrective lenses for comfortable vision and, more particularly, for good near vision. For this, special goggles were obtained which permitted mounting of the prescription lenses be-

hind the proper density absorptive lenses. In addition to those requiring as much as 8 diopters or more of plus or minus correction, there are a few persons who are aphakic. Some individuals require correction for their jobs but need no correction for their off-the-job activities.

for a sterilization and packaging service of these items. More recently, we have been investigating the use of plastic lenses, and as one plastic lens weighs 6.7 grams and one glass lens, 14.4 grams, we may consider quite seriously the use of this new type if the problem of surface abrasion can be cared for.

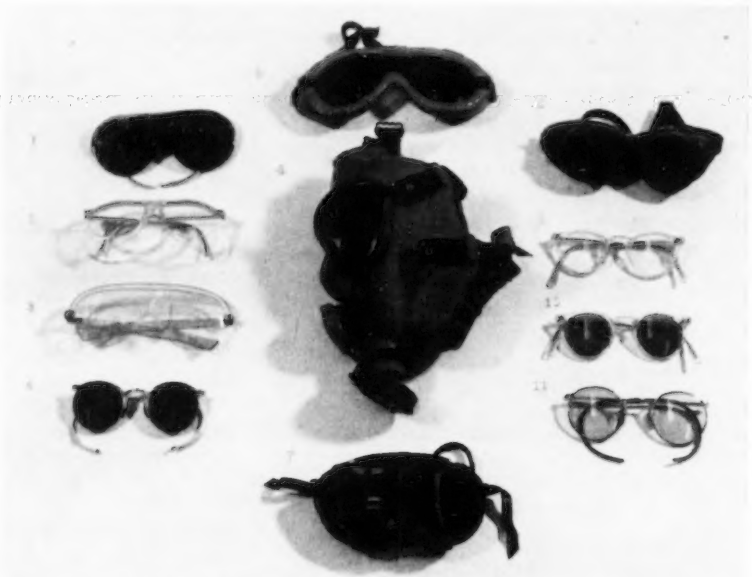

FIG. 4—Types of personal protective eye devices dispensed to Laboratory personnel by the Health Division.

Of the Laboratory employees, approximately 67 per cent wear prescription glasses, either personal or safety. Of this 67 per cent, approximately 75 per cent have prescription safety spectacles. About 80 per cent of the total Laboratory population wear safety glasses and of these, 65 per cent are prescription ground and the remaining 35 per cent are plano. The policy has been always to fit and adjust these plano safety glasses with as much care as the prescription eyewear. These glasses are issued individually, and there is no need

Not all spectacle dispensing is a matter of total simplicity. One worker who wears a high cylindrical correction brought in his glasses to have them adjusted. He is so sensitive to his correction that a rotation of his lenses of only one degree upsets his balanced vision. After checking the lenses for axis in the vertometer and finding them to be exactly right, our dispenser checked then the adjustment and frame alignment. It proved to be that one hinge at the temples was slightly loose, allowing that side of the frame to drop a little lower

than the other, thus rotating the lenses just enough beyond the point of comfort to impair his vision.

All too often for our liking, it is found that an employee will bring in two copies of his prescription, one for the optician to make a pair of dress glasses, and one for the Health Division to use for his safety eyewear. When the safety glasses are dispensed he will complain of a difference between the two pairs of glasses—a difference other than that of weight. In checking it will be found that in the dress glasses the optical centers may be off, the cylinder axis off, the lens powers erroneous, or something similar, which is entirely a result of carelessness on the part of the optician who fabricated the glasses. The number of incorrect pairs has been considerable, and this does not set well with the employee, for while he is comfortable on the job, his away-from-the-work visual activities may be limiting.

Specific Health Education

Believing implicitly in the worth of the tailored-to-fit health education medium, for approximately five years we have been designing and building our own exhibits for display. One of these drawings depicts the issue of occupational eyewear, and the speaker when showing this slide in a talk to employees discusses the types of safety glasses, the maintenance services available, and the need for constant use and up-keep. Frequently the subject of visual health is presented in display format, and on one occasion the need for proper maintenance of safety eyewear was emphasized through use of three dimensional figures in an exhibit with high color-and-design appeal.

In keeping with the concept that industrial health is community health, a column is written for the *Oak Ridge National Laboratory News*, a four-page slick newspaper which covers the news of the week and is mailed to the workers' homes each Friday. The column is read by the wives of the employees, and the subject under discussion often comes within the area of visual well-being. For example, one column (continued a week later) was devoted to strabismus and was headlined by the editor "Psychological Angle Results in Case of Cross Eyed Child." Other columns presented discussions of the functions of various eye specialists, the fallacies regarding certain tinted lenses, and first aid for eye emergencies. This particular medium is read widely, for as one chemist put it, "Doc, I saw your ad about feet, so I brought mine in for you to look at." A pamphlet rack holds publications of all kinds, from bookmarks to comic books, and among these are pamphlets on eyes and vision.

With fair frequency, descriptions of the total health effort are carried to the community as talks to civic clubs, church groups, parent-teacher gatherings, fraternal orders, and boards of social agencies. Always, a review of the occupational vision program is included in the material.

Illumination

Infrequently, unusual problems in illumination arise and consultation is sought either as to the desired type of artificial lighting indicated or the eye equipment required for comfortable vision in areas of high contrast. The matter may involve merely the provision of eccentrically placed additional fluorescent fixtures, tilted to give the maximum needed in foot candles or there may be a question of the optimal lighting in a health physics personnel monitoring laboratory where technicians spend the entire day examining exposed badge meter film in a microscopic field.

On occasions, impaired illumination may be encountered in certain work areas, and complaints will be received

centering about the vague entity of "eyestrain." Measurements of the light intensity are made, and if there is an inadequacy present, recommendations to increase the light source and distribution at the work site are made.

Color

In a modern laboratory where change is a constancy, two features are presented for consideration that involve patternings in color. One concerns merely the scheme to be employed for the general decor of a building, and the other, the compliance with color codes established by national agencies for safe practices in industrial operations. Power tools and machinery are painted in accordance with the recommended codes[2,3] in order to assure a safer operation of hazardous equipment. A certain pride seems to manifest itself in a worker who is operating an immaculately maintained, colorful device.

Within the past few months the Safety Department presented a question concerning the institution of a new type of adhesive-backed label for identification of utility lines. They wished to know if those employees with impaired color discrimination would be able to read the legend. The group known as partially or completely color blind was called in and tested with the samples. All were able to read the markings, and they were put into use without fear of their meaning being lost to certain of the personnel.

The addition of nuclear energy as a health hazard of great dimension—when uncontrolled—has necessitated the introduction of a new color in the code to allow identification of radioactive sources or radioactively contaminated areas. For this, magenta and yellow were selected, and one or the other (or both) identifies many items about the Laboratory: the protective clothing worn, the waste can containing contaminated materials, the trucks used to haul away these items, and the tags that demarcate specific areas. As shown in figure 5, a new figure has been selected for additional identification purposes.

Special Programs

It is of the greatest importance that there be complete documentation of all clinical events in the employment life of the atomic energy worker. This, in our peculiar sort of industry, goes beyond the mere notation in the clinical record of the health happenings and alterations of status that beset the individual. Whereas words can describe a condition with exactitude in definition, not always can they match the detail of photographic recording. Therefore, in addition to the sheets of data in the employee's folder, clinical photographs are made of striking positive findings at the time of pre-placement physical examination, at time of injury or illness, and again, at time of recovery if the somatic changes are grossly demonstrable.

Included in this are photographs of the eye which are taken with a conveniently operated color camera, the simplicity of which permits a photograph to be taken in less than one minute. The transparencies measure 28 mm. by 40 mm. and can be enlarged in a desk model viewer to approximately five diameters. When it is important to record the offending agent causing the injury or to have a remotivating medium for health education purposes, a photograph will be taken, for example, of the broken nonsafety glasses being worn in a hazardous area. A photograph can tell its own story, without employment of the scare technic used with the publication of the sanguineous sagas of the safety supply salesmen.

More recently some research has been undertaken in connection with the cause of hypertension, and we have had occasion to procure a retinal camera for the

photography of the retinas of employees with an elevation of blood pressure. This same unit can be used to take external views of the eye and to follow changes in the lenses of certain workers.

with the slit lamp by a competent ophthalmologist. This aids in the identification of pre-existing lesions unrelated to job exposure and permits a consistent follow-up of any specifically caused lenticular opacities.

FIG. 5—Radiation tag used to distinguish radiation sources or contaminated areas, front and back.

Before the advent of large scale use of atomic energy and its attendant full-range employee protection program, there was a pre-World War II scientific personnel engaged in working with cyclotrons. In a few of this group, morphologically characteristic radiation cataracts have appeared. Every six months, or more frequently when indicated, these researchers along with certain of our current employees are examined

SUMMARY

The visual health maintenance program of one of the nation's largest atomic energy research facilities has been reviewed in its development and found to consist of the following component parts:

1. Analysis of visual demands for specific jobs
2. Identification of unusual eye performance requirements

3. Testing of employees, using a stereoscopic instrument with a complete battery of tests
   a. As part of the preplacement physical examination — tested with and without correcting lenses
   b. At established frequencies determined by visual changes, job assignment, requirements for driver's license, administrative needs (promotion, retirement, etc.) and nature of illness causing absence
   c. At time of injury to the eye and at time of recovery from injury
4. Treatment of eye burns and injuries through use of special equipment
5. Record maintenance of clinical events resulting in visual or anatomic change of the eye
6. Program of procurement, measurement, receipt, verification, dispensing, adjustment, and follow-up care of occupational eyewear
7. Provision of special materials and devices for the more visually impaired
8. Maintenance of a full spectrum of safety eyewear to allow protection in all types of work situations
9. Health education through use of an orientation program, locally devised posters and exhibits, pamphlets, and group talks within the Laboratory and the community
10. Study, recommendation, and follow-up of special illumination needs
11. Consultation in special situations requiring assistance in color selection and use
12. Documentation of pertinent findings in visual health by means of illustrations secured through external color and retinal photography
13. Periodic ophthalmologic examination of employees whose work contact may be potentially productive of lenticular changes

CONCLUSIONS

1. The newest addition to modern industry, the large research laboratory in atomic energy, requires complete consideration of every visual need of the employee.
2. Because of the nature of the operations involved, the maximum in eye correction and protection should be provided in order to guarantee the minimum in sense-loss in a highly skilled and difficult-to-replace personnel.
3. A broad program should include not only the physical armamentarium with which to accomplish the mission but the feeling needed to convey the concept that the health staff is interested in the employee as an individual with pride, worth and dignity and as a contributor to society.

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COMPENSATION PROBLEMS

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COMPENSATION problems in the eye field and technics for estimating the percentage of loss of visual efficiency are even more erratic and complicated than compensation problems for other injuries and loss of members of the body or functions of the body. Because this is especially true, Second Injury Funds were evolved to help provide an answer.

Second Injury Funds serve to cover the much greater sum of money involved when a one-eyed man loses his second eye. In such an event the insurance company has to pay the loss of an entire man; in other words, total disability—not only the loss of an eye. Inasmuch as this is exceedingly expensive, the reluctance of management to risk an increase in insurance rates has been shown in the nonhiring of any one-eyed individual except in states where Second Injury Funds exist. The Second Injury Fund was originally set up as a sum of money collected by a number of industries in a given state which would be drawn on to take care of those cases where a one-eyed man loses his other eye or needs total compensation. Very large sums of money were collected and set up in this fund, and it was not until several years later that it was discovered how very low the actual incidence of such second injuries was and that much more money had been set aside than was necessary. The Second Injury Fund, as far as the author is aware, was started on a private basis in the hope that the idea would be taken up by states.

To meet the challenge presented by war veterans in their pursuit of gainful employment, the idea of Second Injury Funds received even greater impetus. Previously (to 1944) there had been Second Injury Funds in only twenty states: Arkansas, Idaho, Illinois, Maine, Michigan, Missouri, Massachusetts, Minnesota, New York, North Carolina, New Jersey, North Dakota, Ohio, Oklahoma, Oregon, Rhode Island, South Carolina, Utah, West Virginia and Wisconsin. The following states do not have such a fund: Florida, Georgia, Louisiana, New Mexico, Virginia and Nevada. These states are not highly industrialized but there must be occasional cases where such legislation would be of great benefit. Montana enacted such a law in 1950.

The need to utilize all available manpower, and certainly to use all who desire employment, is increasing. The President's Employ the Handicapped Committee has been urging such employment, as have many other organizations. However, before one can properly urge the employment of the physically handicapped, state legislation must, in many instances, be redesigned and redrafted to make this possible. In Wisconsin, for instance, an individual with an arrested case of silicosis, examined and verified by a panel of doctors, is not necessarily turned down by industry. If this person, whose placement is clearly outlined and limited, has a flare-up of his silicosis, industry is not held responsible. In this way, the man is put to work and given a gainful occupation without any risk to anyone including himself. In Michigan, the opposite is true, and therefore industry turns down many patients with arrested cases of tuberculosis and silicosis who could, should and want to work. When it comes to eyes and the employment of men with only one eye, the same situation holds. All too often, industry has no protection, the insurance company has no protection, the man has no job. Actually, there are not only many jobs

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such a man can do easily but there are some jobs that a one-eyed man can do better than a two-eyed man. It has been statistically proved that the physically impaired individual offers less possibility of accident than the so-called normal individual when he is properly placed on a job for which he is physically qualified.

It is important that everyone who has to do with hiring, firing and placement of human beings on any kind of a job at all, whether it is military or civilian, be cognizant of the fact that the objective is always to "match physical capacities to job demands." As Bert Hanman, in his important book,[1] says: "Physical handicap is confused with occupational handicap." When Hanman did his first careful studies he realized that eyes and visual skills were as yet at the bottom of the list in such evaluation. It is true that at that time nothing had been done to prove that eyes have patterns of visual skills just the same as the other physical aptitudes have patterns. Aviation, where it was a life and death matter, was the first industry to investigate anything more than distance acuity before assigning people to jobs. Since 1941, however, the question of job placement on a visual basis has been developed fully. We now know that a complete pattern is recognized for every job; for instance, a crane operator must not only have perfect distance acuity but must have binocular vision, i.e., stereopsis and normal muscle balance, as well as normal color appreciation. An opposite type of pattern would be illustrated by small parts assembly or by critical jobs where perfect *near* vision and normal near muscle balance are needed. This research, stemming from Tiffin's work at Purdue Research Center,[4] made it possible to set patterns for all jobs. Lockheed Aircraft, during the war years, placed as high as three hundred totally blind individuals on highly skilled work (even riveting) and found that these people were not only eager to maintain production but availed themselves of all possible protection and had a lower accident rate than others in the same plant.

The idea of the Second Injury Fund has been carried into other fields of physical disability and is now usually maintained by assessments from industrial insurance carriers. The assessments in most cases are estimated on the basis of the number of disabling and fatal cases sustained by the industry. For instance, in Alabama in cases of death where there are no dependents, the employer or insurer pays $1,000 to the Director of the Department of Industrial Relations within 18 months, or $500 if paid in one year. In Connecticut, the fund is supported by a tax on insurance funds, i.e., 1 per cent of the money paid in compensation by the carrier or employee until the fund is depleted.

It is difficult to classify the provisions and interpretations of the law in various states. Some do have a general pattern; Arizona, Arkansas, California, Colorado, Connecticut, Maine, Maryland, Massachusetts, Michigan, Ohio, Oregon, Pennsylvania, Tennessee, and Wyoming have included special provision for veterans.

In order to properly evaluate the existing legislation, much more experience is needed in the practical application of the law. However, the principle is basic. Such laws are being considered in all states where they seem necessary. In no case is there any consideration of dropping the law itself—only of developing and extending its benefits.

It is possible that Industry is not taking full advantage of the opportunity to use these funds which it is supporting in order to employ handicapped people. A study of some of the records indicate that comparatively few claims have been paid. In 1950, when this study was made, five states reported no claims had

been made on the fund. During a six-year period (1943 through 1949) Missouri has had a total of 509 cases (334 of them concerning eyes). The total amount paid into the fund was $75,130 and the amount disbursed, $25,160.48. Missouri maintains a rehabilitation program in connection with their fund which would naturally increase the cost of individual cases. Alabama had 18 cases in three years with a total outlay of $9,000. In five years, California had about 30 cases, some of which were denied. Indiana paid $35,000 in 1949, one claim amounting to $4,000. In five years, Tennessee had three claims amounting to $13,017.19. Other states report small expenditures.

Waivers

Waivers, on the whole, have been used with unsatisfactory results as very few industrial commissions will pay attention to a waiver clause if a man has actually been disabled in industry. A man cannot, in practice, sign away benefits due him for injury or for a condition aggravated by an industrial hazard. There are states in which the waiver clause has been on the books but no one has had the nerve to test it. Six states have waiver clauses which more or less nullify the benefits to be derived from the Second Injury Fund. These waivers permit the employee to sign a document waiving any benefits for an injury or disease incurred in present employment which might be caused or aggravated by previous disability. Wisconsin, one of the states that lead in the effective handling of compensation claims, permits the employment of men who are totally blind, who have epilepsy or who have certain types of arrested tuberculosis or silicosis. To quote the director of Workman's Compensation in Wisconsin: "We do not favor waiver clauses and believe that waivers by epileptics and totally blind are no longer desirable. That is because when these are properly placed in industry, records indicate that injuries to them are actually at a lesser rate than in the case of normal persons. Proper placement must, of course, be made. When so placed and in the light of the Second Injury provision employers should have little fear as to the additional cost which may be involved under laws properly formulated."

General Factors

The above information, which seems at first glance to be very slim, was actually obtained only after many weeks of searching and correspondence with the State Industrial Commissions, consultation with insurance executives and with industrial physicians. Differences in kinds of compensation set-ups, especially as far as the eyes are concerned, are tremendous. One of the real objectives that should be obtained eventually, and toward which the Pan-American Association of Ophthalmology, the American Academy of Ophthalmology and Otolaryngology and the National Society for the Prevention of Blindness, can add impetus, is some basis of standardization; standardization of the type of information required in reports sent in on each and every industrial case; standardization of the method of evaluating "per cent loss of visual efficiency"; standardization and revision of amount of compensation due for various degrees of "loss of visual efficiency"; and (what is most important) the organizing of a carefully considered study on certain specific types of eye compensation which are now most unrealistic in their approach. A striking example is the case in which a man has sustained a traumatic cataract, has had the cataract removed and has been provided with glasses which give him 20/20 vision in the injured eye.[3] In many states, this person is given 100 per cent loss of visual efficiency in that eye, because it is an aphakic eye (an eye in which the living lens has been removed) which can-

not, by the laws of physics, have more than 20/200 vision without correction in the average patient. Physiologically and anatomically speaking, however, the patient has come through his experience with normal acuity (corrected). The insurance companies feel, quite rightly, that if they have to pay a 100 per cent loss anyhow, they might as well pay it at once *before* the patient is operated upon and they have to pay additional hospital and surgical expenses. This, then, leaves it up to the injured man to find a doctor and pay all of the bills. The added expenses are often very great, cutting down his real and desired benefits.

There was a reason for this decision, however, because the man with one normal eye and one aphakic eye actually is not as well off even with 20/20 corrected vision as he was before he became aphakic. The man who has had a traumatic cataract and has been provided with glasses in order to see normally has sustained a loss of visual efficiency and should have some compensation. But the confusion—double vision, dizziness—that is supposed to follow the removal of a cataract and correction of aphakia when the other eye is normal has been shown by a number of men to be much less frequent than actually anticipated.[3] There are many men working in industry who have had a cataract removed in one eye, who have normal vision in the other eye, and who are wearing a full correction without trouble. Where this is the case, then compensation should be based on all factors. On the other hand, in cases in which the man is unable to wear his full correction (since contact lenses have been available, these cases have been reduced to almost nil), compensation should be evaluated on an entirely different basis. In other words, there should be a sliding scale of compensation. Medical and legal authorities in the field, by a careful study of literature, by knowledge of the function of the eye, by study of insurance companies' experience, can work out and present to the Workman's Compensation Boards in the various states a more realistic solution. When put into legislation, this would make the end result for the injured worker much more fair than it is at present.

The Medical Sub-Committee to the President's Employ the Physically Handicapped Committee is very cognizant of the above and especially cognizant of the state laws that interfere with the employment of one-eyed individuals in industry. It realizes that to blow the trumpets and urge employment of handicapped people *without* working from the bottom up, on those *laws that make it impossible to hire them*, is a very upside-down approach. To urge the impossible always does much more harm than good. The first job is to make it legally possible to hire individuals with certain physical handicaps (one eye) so that neither the employer, nor the employee, nor the insurance company is penalized.

It is important that management be assured that it will only be liable for the disability caused by specific injury incurred while a certain individual was in their employ; that pre-existing physical impairment, whether it be a lung problem or an eye problem which was inactive when the man was hired and in itself not liable to increase, can be figured into the placement of such a person.

The wording of laws in various states is often too flexible or too indefinite in regard to the Second Injury Fund to make the employer feel at ease. It is for this reason that many times these laws do not lend themselves to classification. The wording of other laws leaves the disabled veteran entirely out of the picture as his permanent disabilities have been produced outside of industry.

Because of the detailed and brilliant study of Second Injury Fund laws carried out by Andrew Kalmykow,[2] the author feels that his entire contribution should be part of the printed record.

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President 1952

DETROIT, MICHIGAN

Guest of Honor

FLETCHER D. WOODWARD

Graduate of the University of Virginia Department of Medicine, 1919. Certified by the American Board of Otolaryngology, 1925. At present Clinical Professor of Otolaryngology for the University of Virginia Department of Medicine. A member of the Board of Directors of the American Board of Otolaryngology. For the years 1948 through 1951, a member of the Board of Councillors of the American Academy of Ophthalmology and Otolaryngology. A Fellow of the American College of Surgeons, and a member of the American Otological Society, the American Laryngological Association, the American Laryngological, Rhinological and Otological Society, and the American Bronchoesophagological Association, in which he served as Vice President in 1943. In 1947 he served as Chairman of the Section on Laryngology, Otology and Rhinology of the American Medical Association.

FIFTY-SEVENTH ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

OCTOBER 12 TO 17, 1952

PALMER HOUSE

Chicago, Illinois

OFFICIAL NOTICES

Convention Call

Under the authority of the Constitution and with the approval of the Council, I herewith proclaim that the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology will convene at the Palmer House, Chicago, Illinois, on Monday, October 13, 1952, and will continue through noon of Friday, October 17, 1952.

JAMES MILTON ROBB, M.D.
President

Attest:

WILLIAM L. BENEDICT, M.D.
Executive Secretary-Treasurer

Local Committees

GEORGE E. SHAMBAUGH, JR.
General Chairman

Reception Committee

Lawrence J. Lawson, *Chairman*
Thomas D. Allen
John J. Ballenger
Paul V. Carelli
Cyril V. Crane
Ralph A. Davis
Justin M. Donegan
J. Robert Fitzgerald
Palmer W. Good
George P. Guibor
Paul H. Holinger
William F. Hughes, Jr.
Heinrich G. Kobrak
Peter C. Kronfeld
Francis L. Lederer
Vernon M. Leech
William F. Moncreiff
G. Henry Mundt
Henry B. Perlman
Irving Puntenney
Georgiana D. Theobald
Pierce Theobald
O. E. Van Alyea
Hans Von Leden

Committee on Arrangements

William A. Mann, *Chairman*
Albert H. Andrews, Jr.
Oscar J. Becker
Arthur J. Coombs
Beulah Cushman
John Elsen
Noah Fabricant
Earle B. Fowler
Stanton A. Friedberg
Thomas C. Galloway
Elmer W. Hagens
Myron M. Hipskind
Bertha A. Klien
Robert B. Lewy
John R. Lindsay
G. Henry Mundt, Jr.
Samuel J. Pearlman
Frederic J. Pollock
Samuel Salinger
Gail R. Soper
Walter H. Theobald
Linden J. Wallner
Virgil Wescott
Frank Wojniak

Ladies' Committee

Mrs. G. E. Shambaugh, Jr., *Chairman*
Mrs. Thomas D. Allen
Mrs. Howard Ballenger
Mrs. John J. Ballenger
Mrs. Oscar J. Becker
Mrs. Paul V. Carelli
Mrs. Ralph A. Davis
Mrs. Eugene L. Derlacki
Mrs. J. Robert Fitzgerald
Mrs. Earle B. Fowler
Mrs. Elmer W. Hagens
Mrs. Myron M. Hipskind
Mrs. Paul H. Holinger
Mrs. William F. Hughes, Jr.
Mrs. Heinrich G. Kobrak
Mrs. Lawrence J. Lawson
Mrs. Vernon M. Leech
Mrs. Robert B. Lewy
Mrs. John R. Lindsay
Mrs. William A. Mann
Mrs. G. Henry Mundt
Mrs. G. Henry Mundt, Jr.
Mrs. Henry B. Perlman
Mrs. Gail R. Soper
Mrs. Pierce Theobald
Mrs. O. E. Van Alyea
Mrs. Hans Von Leden
Mrs. Linden J. Wallner

Business Meeting

The Annual Business Meeting of the Academy will be held in the Crystal Room of the Palmer House on Thursday, October 16, 1952, at 5:30 p.m.

The Order of Business:

1. Call to order
2. Report of Executive Secretary-Treasurer
3. Report of the Senior Member of the Council
4. Election of Fellows (See Candidate List, p. 679)
5. Election of Officers
6. Unfinished business
7. New business

All Fellows and candidates for election to Fellowship are urged to attend.

Council Meetings

The Council of the American Academy of Ophthalmology and Otolaryngology will convene at 9 o'clock, Saturday morning, October 11, 1952, in Private Dining Room 4 on the third floor of the Palmer House, Chicago. There will also be a meeting of the Council on Wednesday, October 15, at 12:30 in Private Dining Room 5. Members desiring to be heard or to present business will confer with the Executive Secretary-Treasurer for appointments.

JAMES MILTON ROBB, M.D.
President

New Business

Proposals for new activities often are presented too late during the Academy session to afford time for deliberation. Therefore, new items requiring Council action should be presented in writing to the Executive Secretary-Treasurer early so that the Committee concerned may have an opportunity to study the matter prior to presentation to the Council.

Public Relations

Contributors to the scientific program are requested to send copies of their papers to the Secretary for Public Relations, Erling W. Hansen, M.D., 90 South Ninth St., Minneapolis 2, Minn., at the earliest possible moment.

Exhibits

The Scientific and Technical Exhibits will open at 9 o'clock Sunday morning, October 12. They will close at 4 o'clock Sunday for the President's Reception. From Monday through Friday they will open at 8:30 a.m. and close at 6:00 p.m. except that they will close Friday, October 17, at 12 o'clock.

Dues

Receipt card for 1952 dues is required for registration at the Academy meeting. Those who have not paid dues for 1952 are requested to remit to W. L. Benedict, M.D., Executive Secretary-Treasurer, 100 First Avenue Bldg., Rochester, Minn. In case of loss, a duplicate receipt card may be obtained upon request.

Registration

General registration will begin at 9 o'clock Sunday morning, October 12, in the Foyer to the Grand Ball Room, Fourth Floor.

The regulation badge secured at the time of registration is essential for admittance to all activities. To facilitate registration, the following sections will be provided:

Advance Orders: Members who have reserved instruction course tickets register here. Advance orders are held here for collection.

Members without Advance Orders: Please present Receipt Card for 1952 dues. Dues outstanding may be paid at this section.
(Application blanks for fellowship in the Academy may be obtained here.)

Instructors: All persons presenting instruction courses register here to secure special badge. There is no registration fee for instructors who are not members.

Nonmembers: A registration fee of $5.00 is required of all guests (see next column). The following exceptions are made:

1. Candidates for Academy fellowship whose names appear in the July-August issue of the TRANSACTIONS.
2. Residents in training and registrants for the 1952-1953 Home Study Courses presenting guest cards obtained before the meeting (see below).
3. Guests taking part in the Scientific Program or Exhibits.

Orthoptic Technicians: All orthoptic technicians register here. Members of the American Association of Orthoptic Technicians who have ordered tickets for Orthoptic Technicians Instruction Courses may pick them up here. Tickets will be on sale for those who have not yet purchased them.

Guests

Physicians who are not members are welcome to attend the meeting of the Academy as guests and to participate in all the activities. All guests, however, will be required to register and a fee of $5.00 will be charged.

In the past, a number of Fellows and registered guests of the Academy have indicated that they wish their *office nurses, technicians,* and *similar personnel* to attend parts of the Academy program or exhibits. Such individuals will be allowed to register upon presentation of a written request from a Fellow or if accompanied by a registered guest or Fellow. The guest fee of $5.00 will be required. An exception is made for all members of the American Association of Orthoptic Technicians, who may register without fee.

Residents and 1952-1953 Registrants for Home Study Courses

Men in training in accepted residencies, or those now completing a full academic year (nine months) in basic study, may register without fee upon

presentation of a guest card obtained by written request of their Department Heads.

Registrants who have completed enrollment for the 1952-1953 Home Study Courses in Ophthalmology or Otolaryngology may also request a guest card entitling them to registration without fee.

Requests from Department Heads and Home Study Courses registrants for guest cards should be sent to W. L. Benedict, M.D., 100 First Avenue Bldg., Rochester, Minn., by October 1.

Instruction Course Tickets

The tickets for courses in ophthalmology and the tickets for courses in otolaryngology and maxillofacial surgery will be sold at separate desks in the Registration Headquarters. The bulletin boards behind each desk will indicate what tickets are available. Please consult these boards in making your selections. Bear in mind that individual courses are for one hour only; continuous courses run for two or more hours. Price of tickets is $2.00 per hour.

It is necessary to register and to be wearing regulation badge in order to purchase instruction course tickets.

Ladies' Registration

The wives of members and guests are urged to register at the Ladies' Headquarters in Rooms 15 and 16 on the Club Floor. Registration will open at 9 o'clock, Sunday morning, October 12.

Available Supplies

Collected abstracts of instruction courses, manuals and monographs may be purchased at the supply counter in Registration Headquarters. Guests may place subscriptions to the bimonthly issues of the TRANSACTIONS here, and orders will be taken for the 1952 Bound Volume. Orders will also be taken for prints of the Academy film "Embryology of the Eye" and for related lantern slides and film strips.

Dinner Tickets

Tickets for Alumni Dinners will be on sale at the Registration Headquarters until 2 o'clock Monday afternoon.

Tickets for the Annual Banquet of the Academy, which will be held on Wednesday evening, may be purchased at Registration Headquarters before 2 o'clock on Wednesday afternoon. The price will be $10.00.

Constitutional Changes in Fellowship Status

Life Fellows. Fellows who have paid dues continuously for thirty years will automatically become Life Fellows. They shall not be required to pay any fees or dues or assessments and shall enjoy all the privileges of Active Fellows.

Senior Fellows. Fellows who have paid dues continuously for twenty-five years will be termed Senior Fellows and their dues will be one-half of the dues of Junior Fellows.

from Article V, Section 6, By-Laws

Life Fellows — 1953

Annon, Walter Thomas ....Philadelphia, Pa.
Blassingame, Charles Decatur, Memphis, Tenn.
Bozer, Herrmann Eugene ....Buffalo, N. Y.
Bruner, Abram Breneman ...Cleveland, Ohio
Clerf, Louis Henry .....Philadelphia, Pa.
Dean, Alfred .....Grand Rapids, Mich.
Dorge, Richard Irving ...Minneapolis, Minn.
Gill, G. Bache .....Washington, D. C.
Goldman, Harry G. .....New York, N. Y.
Harris, Robin .....Jackson, Miss.
Hetrick, Llewellyn Evans, Hampton, Conn.
Howard, Lloyd G. ....Council Bluffs, Iowa
Johnson, Thomas Holland ..New York, N. Y.
Lerner, Macy Levi .....Rochester, N. Y.

Looper, Edward Anderson ..Baltimore, Md.
Lyman, Harry W. .....St. Louis, Mo.
McKinney, Alexander R. ....Saginaw, Mich.
Myerson, Mervin Carueth, Beverly Hills, Calif.
Parrish, Robert Eunice ..San Antonio, Texas
Proetz, Arthur Walter .....St. Louis, Mo.
Somberg, Joseph Sheldon ....Oakland, Calif.
Tomassene, Raymond A. ..Wheeling, W. Va.
Townsend, John Ferrars ...Charleston, S. C.
Tucker, Gabriel .....Philadelphia, Pa.
Wendel, Jacob S. .....Detroit, Mich.
Winter, John Arthur .....Duluth, Minn.

Senior Fellows — 1953

Adams, Charles Joseph .....Kokomo, Ind.
Allen, Orris T. .....Terre Haute, Ind.
Allen, Thomas Dyer .....Chicago, Ill.
Alvis, Bennett Y. .....St. Louis, Mo.
Blair, Mortimer Warren ....Philadelphia, Pa.
Brownell, Morton Emmons ....Wichita, Kan.
Bryan, James Leslie .....Nashville, Tenn.
Bulson, Eugene Loring .....Ft. Wayne, Ind.
Carruth, Howard Ernest .....Portland, Ore.
Cassady, J. Vernal .....South Bend, Ind.
Christoph, Carl H. .....Chicago, Ill.
Cody, Claude Carr, Jr. .....Houston, Texas
Cowan, Alfred .....Philadelphia, Pa.
Crumbine, Norman R. .....Beaver, Pa.
Cunning, Daniel S. .....New York, N. Y.
Davis, John Calvin, Jr. .....Omaha, Neb.
Deal, John Francis .....Springfield, Ill.
DeWitt, Edward Nicholas ..Bridgeport, Conn.
Dixon, Otto Jason .....Kansas City, Mo.
Eber, Carl Theodore .....St. Louis, Mo.
Eimer, Charles Eugene .....St. Louis, Mo.
Ellis, C. Ward .....West Olive, Mich.
Fernlund, George Byron ...New York, N. Y.
Folken, Frank Garrett ....Albert Lea, Minn.
Ford, Hanby Lewis .....Champaign, Ill.
Fuchs, Valentine Henry ...New Orleans, La.
Gaudet, Lucien Sydney .....Natchez, Miss.
Glick, Harry Naylor .....St. Louis, Mo.
Glowacki, Ben Francis .....Detroit, Mich.
Goldman, A. Milton .....Rockville Centre, L. I., N. Y.
Greene, Laurence Whitridge ..Denver, Colo.
Greenfield, William John ..Hackensack, N. J.
Hale, Arthur Stanley .....Detroit, Mich.
Hall, Edwin Perry .....Oneonta, N. Y.
Hardy, LeGrand Haven ....New York, N. Y.
Harrison, Fred Hazard....Connellsville, Pa.
Hill, Frederick Thayer ....Waterville, Maine
Hill, Howard Foster .....Waterville, Maine
Howard, Harvey James ....Clearwater, Fla.
Hughes, Ray Whitbeck ....Lake Worth, Fla.
Jackson, Atras Earl .....Ft. Worth, Texas
Jones, Cecil Cain .....Des Moines, Iowa
Jones, Edley Hicks .....Vicksburg, Miss.
Jones, Vincent L. .....St. Louis, Mo.
Karkeet, Robert B. .....Portland, Ore.
Kelley, Isaac D., Jr. .....St. Louis, Mo.
Kelly, Joseph Dominic ....New York, N. Y.
Kennedy, Eldred Weston ..Rochester, N. Y.
Kincannon, LeRoy Taylor ..Birmingham, Ala.
Koenig, Ivan J. .....Buffalo, N. Y.
Krimsky, Joseph .....Hunnington, W. Va.
Kuhn, Hugh A. .....Hammond, Ind.
Lawwill, Stewart .....Chattanooga, Tenn.
Lewis, Philip Meriwether ..Memphis, Tenn.
Lierle, Dean M. .....Iowa City, Iowa
Lucas, Harold Roy .....Portland, Ore.
Lupton, Irving Martin .....Portland, Ore.
Mahoney, Paul Leo .....Little Rock, Ark.
Mason, Roy Earl .....St. Louis, Mo.
Masters, Robert John .....Indianapolis, Ind.
McKee, Joseph Wallace ....Kansas City, Mo.
McNally, William James, Montreal, Que., Can.
Merkel, Charles Carson, Groose Pointe, Mich.
Meyer, Samuel James .....Chicago, Ill.
Montgomery, Daniel Cameron ....Greenville, Miss.
Moriarty, Richard W. ..White Plains, N. Y.
Palmer, Bascom Headen .....Miami, Fla.
Palmer, Frank Ernest .....Sterling, Colo.
Plumer, John Shaffer .....Pittsburgh, Pa.
Powell, Lyle Stephenson ....San Diego, Calif.
Randall, Hiram .....Binghamton, N. Y.
Reese, Algernon B. .....New York, N. Y.
Rhode, Solon Lafayette, Santa Barbara, Calif.
Risser, Edward D. .....Pasadena, Calif.
Rychener, Ralph Orlando ....Memphis, Tenn.
Sanor, Daniel Galbreath ....Columbus, Ohio
Savery, Charles Edward ....South Bend, Ind.
Sinamark, Andrew .....Hibbing, Minn.
Spaeth, Edmund B. .....Philadelphia, Pa.
Spencer, George Albert ..Pacific Grove, Calif.
Sproule, Ralph P. .....Milwaukee, Wis.
Steele, Willard .....Chattanooga, Tenn.
Stinson, William David .....Memphis, Tenn.
Swab, Charles Marion .....Omaha, Neb.
Teall, Raymond Edwin ....Kansas City, Mo.
Thompson, Howard E. .....Dubuque, Iowa
Warren, William C., Jr. .....Atlanta, Ga.
Weber, Henry Charles .....Drexel Hill, Pa.
Whalen, Edward Joseph ....Hartford, Conn.
Willson, Wesley W., North Hollywood, Calif.
Windham, Robert Edward, San Angelo, Texas
Wood, Earl LeRoy .....Newark, N. J.
Woodson, Burbank Palmer ....Austin, Texas
Zubak, Matthew Francis ..Wheeling, W. Va.

CONVENTION ACTIVITIES

PRESIDENT'S RECEPTION

Grand Ball Room

Sunday, 4:30 p.m.

President James Milton Robb and Mrs. Robb invite all members and guests of the Academy to a reception honoring Dr. Fletcher D. Woodward and officers of the Academy on Sunday, October 12, at 4:30 p.m. in the Grand Ball Room.

JACKSON MEMORIAL LECTURE

Sponsored by the Ophthalmic Publishing Co.

Dr. David G. Cogan, Howe Laboratory of Ophthalmology, Harvard University Medical School and Massachusetts Eye and Ear Infirmary, will deliver the Jackson Memorial Lecture at the scientific session of the Section on Ophthalmology, Tuesday morning, October 14. The subject of his paper will be "Congenital Ocular Apraxia."

WHERRY MEMORIAL LECTURE

Sponsored by the Wherry Memorial Fund

The 1952 Wherry Memorial Lecture will be delivered at the scientific session of the Section on Otolaryngology, Thursday afternoon, October 16, by Dr. John R. Lindsay of the University of Chicago. The subject of his address will be "Some Experimental Observations Bearing on Meniere's Disease."

ANNUAL ACADEMY DINNER

Grand Ball Room

Wednesday, 7:00 p.m.

Dress Optional

Capt. Eddie Rickenbacker, President and General Manager of Eastern Air Lines, will address the guests on the subject, "It's Not Too Late."

Following the address an orchestra will furnish music for dancing.

FACULTY LUNCHEON

Private Dining Room 14

Thursday, 12:45 p.m.

President James Milton Robb will be host to all those presenting scientific papers, motion pictures, scientific exhibits, and courses of instruction.

ALUMNI DINNERS

The following alumni groups will hold dinners on Monday evening, October 13, at 6:30 p.m.: (A complete listing including place of dinner and address of the secretary for each group will appear in the Pocket Program issued at the Academy meeting.)

University of Alabama Alumni—Ophthalmology
Barnhill-McCaskey Alumni
Billings Hospital Alumni Association
Boston City Hospital Alumni
Brooklyn Eye and Ear Alumni Association
Dr. J. MacKenzie Brown Alumni
University of Chicago Ophthalmological Alumni Club
Cook County Hospital Alumni
Emory University Alumni
Henry Ford Hospital Alumni

Gill Memorial Eye, Ear and Throat Hospital Alumni—Annual Spring Congress
Dr. John F. Gipner Alumni
Harper Hospital Alumni
Otolaryngologic Alumni Association of the University of Illinois and the Illinois Eye and Ear Infirmary
Illinois Eye and Ear Infirmary Alumni
Dr. Chevalier Jackson Alumni
Jefferson Hospital Otolaryngological Alumni
Dr. John R. Lindsay Alumni
Massachusetts Eye and Ear Infirmary Alumni—Ophthalmology
Massachusetts Eye and Ear Infirmary and Harvard Medical School Alumni—Otolaryngology
Mayo Clinic Alumni
Minnesota Alumni Association
Mount Sinai Hospital of New York Alumni
University of Nebraska College of Medicine Alumni—Ophthalmology
New Orleans Eye, Ear, Nose and Throat Hospital — Charity Hospital Alumni
New York Eye and Ear Infirmary Alumni
New York University, New York Post Graduate School and Bellevue Hospital Alumni
Walter R. Parker-Roy Bishop Canfield Alumni
University of Pennsylvania and Wills Hospital Alumni—Ophthalmology
University of Pennsylvania Alumni—Otolaryngology
Pittsburgh Eye and Ear Group
Presbyterian Hospital Institute of Ophthalmology Alumni
Presbyterian Hospital (New York) — Ear, Nose and Throat Group
St. Luke's Hospital Alumni
Stanford Eye, Ear, Nose and Throat Alumni
University of Toronto Eye Alumni
Washington University Alumni—Ophthalmology
Washington University Alumni—Otolaryngology
Wilmer Institute Alumni—Johns Hopkins Hospital

LOCATION OF ROOMS
SCHEDULED FOR ACADEMY ACTIVITIES

THIRD FLOOR	FOURTH FLOOR	CLUB FLOOR
P.D.R. 1 through 9	Exhibit Hall	P.D.R. 14 through 18
P.D.R. 11	Red Lacquer Room	
Crystal Room	Foyer to Grand Ball Room	
	Grand Ball Room	
	Executive Office	
	Public Relations Office	

Palmer House
Chicago

P.D.R.—Private Dining Room

Club Floor may be reached by stairway from Fourth Floor

CANDIDATES--1952

The following candidates have qualified for Fellowship in the American Academy of Ophthalmology and Otolaryngology and will be voted upon at the Annual Business Meeting, Thursday, October 16, 1952. Objections to the election of a candidate must be filed with the Executive Secretary-Treasurer previous to Saturday, October 11, 1952.

CANDIDATES CERTIFICATED BY BOARDS

Ophthalmology (OP)
Otolaryngology (ALR)

Abbey, Joseph Ascher, 429 Palmero St., Corpus Christi, Texas, ALR

Abraham, Raymond Arthur (Capt.), Eye Clinic, U. S. Army Hospital, Camp Roberts, Calif., OP

Adelman, Nathan, 123 E. 83rd St., New York 28, N. Y., ALR

Ajello, Dominick Arnold, 1 Nevins St., Brooklyn 17, N. Y., OP

Alexander, Robert L., Cleveland Clinic Foundation, Cleveland, Ohio, OP

Allen, Wallace Edgar (Comdr.), U. S. Naval Hospital, Oakland 14, Calif., ALR

Alter, Albert Jerviss, Medical Center Bldg., 424 W. Woodruff Ave., Toledo 2, Ohio, OP

Anderson, William Clayton, Jr., 212 W. Boscawen St., Winchester, Va., OP

Arnold, Ira Lee, Jr., 535 McCallie Ave., Chattanooga 2, Tenn., OP

Attridge, Frank Roy, 2525 Pine St., Vancouver 9, B. C., Canada, ALR

Ayash, John Joseph, McCannel Clinic, 21 Central Ave. West, Minot, N. D., ALR

Babacz, Teofil, 450 Gay St., Phoenixville, Pa., OP

Bagby, Richard A., 509 Bay St., Tampa, Fla., ALR

Bain, James Britton, 1712 Rhode Island Ave. N.W., Washington 6, D. C., OP

Barrett, Sterling Archie, 721 Black Bldg., Waterloo, Iowa, ALR

Bartley, Max Donald, 803 Hume Mansur Bldg., Indianapolis 4, Ind., OP

Baruch, Richard, 22 Hamilton Ave., Yonkers 5, N. Y., OP

Battles, Morris Lyle, 370 E. Town St., Columbus 15, Ohio, OP

Baum, Gilbert, 235 Columbus Ave., Port Chester, N. Y., OP

Bedrossian, E. Howard, 2027 Spruce St., Philadelphia, Pa., OP

Bell, Bernard Thomas, 148 First St. North, St. Petersburg 1, Fla., OP

Bell, Houston Lesher, 711 S. Jefferson St., Roanoke, Va., ALR

Bell, Richard Parker, Jr., 11621 Detroit Ave., Cleveland 2, Ohio, OP

Berger, John Van Eman, Jr., 224 W. Washington Ave., Madison 3, Wis., OP

Bernfeld, Michael, 123 S. Stone Ave., Tucson, Ariz., OP

Billet, Edwin, 2625 Grand Concourse, Bronx 68, N. Y., OP

Binder, Donald Keever, 708 Union St., Schenectady 5, N. Y., OP

Birenboim, Irvin Myron, 530 Professional Bldg., 1103 Grand Ave., Kansas City 6, Mo., ALR

Blair, Frank Wilkinson, 369 Green Lane, Philadelphia 28, Pa., OP

Boggess, Julian Eugene, 911 College St., Columbus, Miss., ALR

Boyd, Charles Glenn, 100 W. 59th St., New York 19, N. Y., OP

Boyd, James Leslie, 100 W. 59th St., New York 19, N. Y., OP

Boyers, Sidney Steven, 413 60th St., West New York, N. J., ALR

Branca, Henry Edward, 210 Arcade Bldg., Fort Pierce, Fla., OALR

Breakstone, Edgar O., 11200 S. Michigan Ave., Chicago 28, Ill., ALR

Brenin, Goodwin M., 3 E. 66th St., New York 21, N. Y., OP

Bricca, Constantine R., Jr., 909 Hyde St., San Francisco 9, Calif., OP

Brickley, Paul M., Mayo Clinic, Rochester, Minn., OP

Brown, Russell Nutt, 870 Fidelity Medical Bldg., Dayton 2, Ohio, OP

Bruce, Robert Addison, 60 Wyoming St., Dayton 9, Ohio, OP

Bryce, Douglas P., 222 Medical Arts Bldg., Toronto, Ont., Canada, ALR

Buck, Leonard S., 130 N. La Cienega Blvd., Los Angeles 48, Calif., ALR

Bunting, Williston Penfield, 1622 Professional Bldg., Kansas City 6, Mo., ALR

Burch, George William, 557 Fair Foundation Bldg., Tyler, Texas, OP

Buxton, Jorge Norman, 17 E. 72nd St., New York 21, N. Y., OP

Caldwell, Pearson Cash, 506 Goodhue Bldg., Beaumont, Texas, OP
Cales, Kenneth Jackson, 233 A St., San Diego 1, Calif., ALR
Cameron, John Hugh, Northwest Clinic, 220 S. Broadway, Crookston, Minn., ALR
Campbell, John Edgar, Jr. (Capt.), U.S. A.F. Hospital, MacDill Air Force Base, Fla., OP
Cardona, John Charles, 75 N. Broadway, Nyack, N. Y., ALR
Carter, Harold G., 3344 Fourth Ave., San Diego 3, Calif., ALR
Casterline, Donald Graham, 2316 Dwight Way, Berkeley 4, Calif., ALR
Cayce, Lee Farrar, 713 Bennie-Dillon Bldg., Nashville 3, Tenn., ALR
Chambers, Benjamin Morris, 615-16 Grant Bldg., 44 Broad St. N.W., Atlanta, Ga., OP
Chapman, Jules Benjamin (Lt. Col.), 80th Medical Group, APO 30, c/o Postmaster, New York, N. Y., OP
Cholst, Mortimer R., 1 Nevins St., Brooklyn 17, N. Y., OP
Clark, Graham, 635 W. 165th St., New York 32, N. Y., OP
Clower, James Wesley, 131 Broadway, Daytona Beach, Fla., OP
Cobots, Joseph Charles, 423 E. Ninth St., Chester, Pa., OP
Cogswell, Thomas G., 46 Pleasant St., Concord, N. H., OP
Collins, Raymond C., 38 Washington St., East Orange, N. J., OP
Costner, Alfred Nixon, Duke Hospital, Durham, N. C., OP
Cronin, John J., Jr., 414 S. Oak Park Ave., Oak Park, Ill., ALR
Cronin, Thomas P., 101 Bay State Rd., Boston 15, Mass., OP
Cross, Richard Joseph (Capt.), School of Aviation Medicine, Randolph Field, Texas, ALR
Cummings, George Otis, Jr., 47 Deering St., Portland 4, Maine, ALR
Cunin, Harry L., 1801 Walnut St., Allentown, Pa., ALR

D'Ambrosio, Francis Anthony, 520 Commonwealth Ave., Boston 15, Mass., OP
Davis, Frederick Jefferson, 224 W. Washington Ave., Madison 3, Wis., OP
Day, Robert McClelland, 635 W. 165th St., New York 32, N. Y., OP
Diamond, Stanley, 536 Mason St., San Francisco 2, Calif., OP
Dickerman, Frederick A., 1138 Elm St., Manchester, N. H., OP
Dickey, Robert Lewis, 401 S. Central Ave., Laurel, Del., OP
Dickinson, Thomas Gilford, 1930 Chestnut St., Philadelphia 3, Pa., OP
Di Francesco, John George, 8314 Fourth Ave., Brooklyn 9, N. Y., OP
Dimling, Carson Simon, 513 Jenkins Bldg., Pittsburgh 22, Pa., ALR
Dimond, Edgar A., 2003 Bryant Bldg., Kansas City 6, Mo., OP
Dismukes, Henry Moseley, 601 Merchants Nat'l Bank Bldg., Mobile 11, Ala., OP
Dixon, Joseph Moore, 4337 Cliff Rd., Birmingham 5, Ala., OP
Docter, Luebert, 26 Sheldon Ave., Grand Rapids 2, Mich., OP
Dodson, John Walter, Jr., 602 Medical Arts Bldg., Hot Springs, Ark., OP
Donnelly, William Allen, 689 Asylum Ave., Hartford, Conn., ALR
Dougan, Archie Fea, 114 E. Broadway, Enid, Okla., OP
Douglas, Robert Edward (Comdr.), U. S. Naval Hospital, Great Lakes, Ill., ALR
Douvas, Nicholas George, 400 Michigan Nat'l Bank Bldg., Port Huron, Mich., OP
Duel, Arthur Baldwin, Jr., 16 Park Ave., New York 16, N. Y., OP
Duggan, John Winston, Wells Pavilion, University of Alberta Hospital, Edmonton, Alta., Canada, OP
Dunn, Paul P., 151 Rock St., Fall River, Mass., OP
Durham, Davis Godfrey, Medical Arts Bldg., Wilmington 1, Del., OP

Earl, Donald H., 804 S. Pacific Ave., San Pedro, Calif., ALR
Edwards, John E. (Major), 121st Evacuation Hospital, APO 301, c/o Postmaster, San Francisco, Calif., OP
Enns, James H., 210 S. Pine St., Newton, Kan., OP
Errgong, William Henry, Jr., U. S. Public Health Hospital, Boston (Brighton), Mass., OP

Failla, John Anthony, 474 Main St., Greenfield, Mass., ALR
Farber, David N., 308 N. Fifth St., Reading, Pa., OP
Fasanella, Rocke M., 840 Howard Ave., New Haven, Conn., OP
Feamster, Robert Cantrell, 13½ E. Choctaw, McAlester, Okla., OP
Feiman, Lawrence H., 425 E. Wisconsin Ave., Milwaukee 2, Wis., OP
Feinberg, Harry, 964 Avenue C, Bayonne, N. J., ALR

Finerman, Wilmore B., 727 W. Seventh St., Los Angeles 17, Calif., ALR
Finton, Max Arthur, 252 E. Lovell St., Kalamazoo, Mich., OP
Fischer, George Gotthardt, 1167 Wilmette Ave., Wilmette, Ill., ALR
Fishler, Harry W., 913 W. Fifth St., Los Angeles 17, Calif., ALR
Floyd, Paul Elliott, 2 Middle St., Farmington, Maine, OP
Fogelhut, Sam, 515 School St., Pittsburg, Calif., ALR
Fox, Joseph Robert, 42 S. State St., Dover, Del., OALR
Fox, Leonard, 3152 Biddle Ave., Wyandotte, Mich., OP
Fox, Ralph McIntosh, 205 Wabeek Bldg., Birmingham, Mich., OP
Frell, Albert Calabria, Gailey Eye Clinic, Bloomington, Ill., OP

Gabriels, Joseph A. C., 374 Hudson Ave., Albany 10, N. Y., OP
Garfin, Samuel William, 371 Commonwealth Ave., Boston 15, Mass., ALR
Gottelfinger, Ralph A., 321 W. Broadway, Louisville 2, Ky., OP
Giannasio, Joseph, 2630 Boulevard, Jersey City 6, N. J., OP
Gifford, Byron Lee, 317 W. Pueblo St., Santa Barbara, Calif., OP
Giles, Julian Wheatley, Box 26, Veterans Administration Hospital, Tuskegee, Ala., ALR
Goldberg, Harry, 90-19 153rd St., Jamaica 2, N. Y., OP
Goodman, William Eric, 167 College St., Toronto, Ont., Canada, ALR
Goodnow, Chester Lloyd, 148 First St. North, St. Petersburg 1, Fla., ALR
Goodstein, Seymour, 197-05 Hillside Ave., Hollis 7, N. Y., OP
Goodwin, Olin Mansell, University Hospitals, Iowa City, Iowa, ALR
Goodwin, Ralph Augustus, Jr., 33 Court St., Auburn, Maine, OP
Gostin, Seymour Bernard, Veterans Administration Hospital, McKinney, Texas, OP
Graeser, Henrik Sofas, 507 Main St., Woodland, Calif., OP
Graham, Walter Raleigh, 1012 Kings Drive, Charlotte 7, N. C., OP
Grant, Charles P., 903 S. 21st St., Birmingham, Ala., OP
Gray, Charles Lucien, University Hospitals, Iowa City, Iowa, ALR
Griesemer, Laurence Crane, 1145 E. Jersey St., Elizabeth 4, N. J., OP
Groom, Joseph J., 624 DuPont Bldg., Miami 32, Fla., OP
Gunzburg, Julian, 193 N. Robertson Blvd., Beverly Hills, Calif., OP

Hadley, Richard B., Highland Hall, Rye, N. Y., ALR
Hamilton, Quentin Paul, 13836 Woodward Ave., Detroit 3, Mich., ALR
Hammerman, Henry Jerome, 2100 Bedford Ave., Brooklyn 26, N. Y., ALR
Hardesty, Hiram Haines, 10515 Carnegie Ave., Cleveland 6, Ohio, OP
Hargrove, Carey Jefferson, 1039 Mellie Esperson Bldg., Houston 2, Texas, OP
Hart, William Milton, 3432 N. Broad St., Philadelphia 40, Pa., OP
Hart, William Ward, 310 Medical Arts Bldg., Shreveport 45, La., ALR
Harvey, Salmon C., 286 Union Ave., Brooklyn 11, N. Y., ALR
Hedges, Thomas Reed, Jr. (Capt.), William Beaumont Army Hospital, El Paso, Texas, OP
Henry, Randall William, 971 Paulsen Medical Dental Bldg., Spokane, Wash., OP
Hesdorffer, Eugene, 1020 N. State St., Jackson, Miss., ALR
Hirsch, Lawrence S., 42 Lincoln St., Framingham, Mass., ALR
Holland, Ned Wilson, 1905 Grand Central Ave., Tampa 6, Fla., ALR
Holmes, Verner S., 210 N. Front St., McComb, Miss., ALR
Holmes, Wendell B., 210 N. Front St., McComb, Miss., OP
Hooper, Kendall, 714 Beach St., Flint 3, Mich., ALR
Hosmer, Matthew N., 384 Post St., San Francisco 8, Calif., ALR
Houston, William Robert, 218 Second Ave., New York 3, N. Y., OP
Irvine, Paul Clarke, 1893 Sheridan Rd., Highland Park, Ill., OP
Jacobius, Henry F., 222 Spring Garden St., Easton, Pa., OP
Jacobson, Payson Bernard, 59 Deering St., Portland 4, Me., OP
Johansen, Vincent E., 909 Hyde St., San Francisco 9, Calif., ALR
Kahan, Edmund, 400 West End Ave., New York 24, N. Y., OP
Kahn, Paul, Jr., 9730 Wilshire Blvd., Beverly Hills, Calif., OP
Kasper, Richard Leon, (Capt.) U.S. Army Hospital, Fort Bragg, N. C., OP
Kasser, Max David, 37 S. 20th St., Philadelphia 3, Pa., OP
Kaye, Raymond John, 301 S. Illinois St., Belleville, Ill., OP

Keeney, Arthur Hall, 1103 Heyburn Bldg., Louisville 2, Ky., OP
Keiber, Henry Frederick, Coker Bldg., Winter Haven, Fla., ALR
Keil, Francis Conrad, Jr., 660 Madison Ave., New York 21, N. Y., OP
Keller, A. Paul, Jr., 1010 Prince Ave., Athens, Ga., ALR
Kennedy, John Anthony, 129 Clinton St., Watertown, N. Y., OP
Kennerdell, Edward Harold, 324 E. Sixth Ave., Tarentum, Pa., OP
Kiess, Robert David, 1838 Parkwood Ave., Toledo 2, Ohio, OP
Killian, Edgar William, 211 S. Third St., Logansport, Ind., ALR
Kimura, Samuel J., University of California Hospital, San Francisco 22, Calif., OP
King, Norman Ellwood (Lt. Col.), Army and Navy Hospital, Hot Springs, Ark., ALR
Kirby, Thomas Joseph, Jr., 102 Second Ave., S.W., Rochester, Minn., OP
Kirk, Robert C., 15644 Madison Ave., Lakewood 7, Ohio, OP
Klabenes, Frank Joseph, 1020 Medical Arts Bldg., Omaha 2, Neb., ALR
Klaiber, John Jefferson, 468 Delaware Ave., Buffalo 2, N. Y., ALR
Klestadt, Walter D., 38 Rock St., Fall River, Mass., ALR
Kline, Oram R., Jr., 514 Cooper St., Camden, N. J., OP
Kogut, Henry Vincent, 1026 Park Ave., Bridgeport, Conn., OP
Kohn, Howard D., 1148 Euclid Ave., Cleveland 15, Ohio, OP
Korsmo, Carl T., 301 Ohio Bldg., Akron, Ohio, ALR
Kottler, Saul, 1214 Nat'l City Bank Bldg., Cleveland 14, Ohio, OP
Kraus, Ralph N., 3310th Medical Group, Scott Air Force Base, Ill., ALR
Kudolla, Charles Rudolph, 815 S. Center St., Casper, Wyo., ALR
Kuehnle, Gustave Russell, 1200 Main St., Dubuque, Iowa, ALR
Kulczycki, Edward (Comdr.), U. S. Naval Hospital, Charleston, S. C., OP
Kundert, Karl F., 1114 Marsh St., San Luis Obispo, Calif., OP
Kuppinger, John Canniff, 613 W. Filmore St., Harlingen, Texas, OP
Kutsunai, Toshio, 272 Kinoole St., Hilo, Hawaii, OP
Lane, Joseph Alan, 912 Provident Bank Bldg., Cincinnati 2, Ohio, ALR
Lasky, Marvin I., 1705 Second Ave., Rock Island, Ill., ALR
Leachman, John William, 26 Clinton St., Warsaw, N. Y., ALR
Lebow, Erwin Edward, 947 W. Eighth St., Los Angeles 17, Calif., OP
Legett, Carey, Jr., 1707 Colorado St., Austin 1, Texas, OP
Leggett, Albert E., Jr., 614 Fincastle Bldg., Louisville 2, Ky., OP
Lehman, Robert N. (Lt. Col.), Murphy Army Hospital, Waltham 54, Mass., OP
Lehner, Robert H., 310 Fifth St., Racine, Wis., OP
Levatin, Paul, 280 MacArthur Blvd., Oakland 11, Calif., OP
Lewin, Wilbur Howard, 906 Olive St., St. Louis 1, Mo., OP
Lewis, John Sidney, 901 Lexington Ave., New York 21, N. Y., ALR
Lide, Lanneau D., Jr., 161 W. Cheves St., Florence, S. C., OP
Lindberg, Winston R., 600 Physicians & Surgeons Bldg., Minneapolis 2, Minn., OP
Livingston, Wiley Kemp, 811-15 Woodward Bldg., Birmingham 3, Ala., OP
Loeb, Donald Robert, 450 Sutter St., San Francisco 8, Calif., OP
Loewy, Arthur, 30 N. Michigan Ave., Chicago 2, Ill., ALR
Loftis, John Riley, Jr., 210 Mobberly Ave., Longview, Texas, OP
Lore, Harry Elmer, Jr., 602 W. University Ave., Urbana, Ill., ALR
Lossef, Samuel, 216 E. 15th St., New York 3, N. Y., OP
Lubchansky, Jacob Harris, 302 State St., New London, Conn., ALR
Lutman, Frank C., 100 W. Coulter St., Philadelphia 44, Pa., OP

Magielski, John E., University Hospital, Ann Arbor, Mich., ALR
Maher, Walter Joseph, 440 E. 20th St., New York 9, N. Y., OP
Maislen, Sidney Earl, 1570 Main St., Springfield 3, Mass., OP
Makart, Carl D., 135 S. La Salle St., Chicago 3, Ill., ALR
Makley, Torrence A., Jr., 150 E. Broad St., Columbus 15, Ohio, OP
Manchester, Thomas, 19-A, Inwood Circle, Atlanta, Ga., OP
Manning, Ephraim Lester, 1820 W. Third St., Davenport, Iowa, ALR
Martin, Herbert, 210 Wall St., Kingston, N. Y., ALR
Martin, John T. (Lt. Col.), Army and Navy Hospital, Hot Springs, Ark., OP
Mather, Robert W., Temple University Hospital, Philadelphia 40, Pa., OP

Mayer, James T., 952 Hanna Bldg., Cleveland 15, Ohio, OP
McClure, Coye W., 1005 Medical Arts Bldg., Oklahoma City, Okla., OP
McCoy, Edward Gordon, 490 Post St., San Francisco 2, Calif., ALR
McGannon, William James, 14597 Madison Ave., Lakewood 7, Ohio, OP
McHugh, Hollie E., 3448 Peel St., Montreal, Que., Canada, ALR
McKenna, John Francis, 711 D St., San Rafael, Calif., OP
McKinney, Worthy William (Major), U. S. Army Hospital, West Point, N. Y., OP
McLoone, John J., 605 W. Montebello, Phoenix, Ariz., ALR
McMahon, Robert John, 277 Alexander St., Rochester 7, N. Y., ALR
McStravog, Lawrence Joseph, 6910 Market St., Upper Darby, Pa., ALR
Medine, Myer M., 57 W. 57th St., New York 19, N. Y., OP
Medwick, Joseph Xavier, 231 N. Duke St., Lancaster, Pa., ALR
Meissner, Tom O. W., 1010 Prince Ave., Athens, Ga., OP
Menger, Harold Charles, 259 Brevoort St., Kew Gardens 15, N. Y., ALR
Michaelson, Jesse Joseph, Medical Bldg., 165 N. Village Ave., Rockville Centre, N. Y., OP
Miles, Paul Wendell, 640 S. Kingshighway, St. Louis 10, Mo., OP
Miller, George Edward, 207 E. Watauga Ave., Johnson City, Tenn., OP
Miller, William A. (Col.), U. S. Marine Hospital, Norfolk, Va., ALR
Mims, James Luther, Jr., 705 E. Houston St., San Antonio 5, Texas, OP
Mohlenbrock, William Dean, 108 N. 14th St., Murphysboro, Ill., ALR
Monson, Keyne P., 1239 B St., Hayward, Calif., ALR
Montreuil, Fernand, c/o Notre Dame Hospital, Montreal, Que., Canada, ALR
Morgan, Morton B., 605 Lincoln Rd., Miami Beach 39, Fla., OP
Mortensen, Andreas V. N., 458 Government St., Mobile, Ala., OP
Moulton, Olin Cates, 130 N. Virginia St., Reno 1, Nev., OP
Muenzner, Richard John, 922 Bankers Bldg., Milwaukee 2, Wis., OP
Murry, Charles M., 512 Van Buren Ave., Oxford, Miss., ALR

Nadbath, Rudolph Paul (Lt. Comdr.), U. S. Naval Hospital, Philadelphia, Pa., OP
Nadeau, E. George, 128 E. Walnut St., Green Bay, Wis., OP
Naquin, Howard A., Johns Hopkins Hospital, Baltimore 5, Md., OP
Norton, Herman Judson, Jr., 277 Alexander St., Rochester 7, N. Y., OP
Nugent, Milton E., 221 Fifth St., Bismarck, N. D., OP

O'Connell, Thomas Leo, 359 Broad St., Providence, R. I., ALR
O'Connor, John William, 1820 Gilpin St., Denver 2, Colo., OP
Odess, John Samuel, 1925 15th Ave. South, Birmingham, Ala., ALR
Ojers, Gaylord Wareham, 313 S. 17th St., Philadelphia 3, Pa., OP
O'Kane, Thomas Walker, 830 Lowry Medical Arts Bldg., St. Paul 2, Minn. OP
Oppenheim, Heinz, Veterans Administration Hospital, Louisville 2, Ky., ALR

Panter, Edward George, 510 Republic Bldg., Denver 2, Colo., OP
Parker, John T., 4800 E. Gibson Ave., Albuquerque, N. M., OP
Parkes, Morey Leonard, 639 W. Ninth St., San Pedro, Calif., ALR
Pearson, Rudolph W., 300 Thayer St., Providence 6, R. I., ALR
Pennington, Glenn Walton, 1318 Scott Ave., Charlotte, N. C., ALR
Peterson, John W., 211½ S. Illinois St., Carbondale, Ill., OP
Peterson, Paul Leland, 702 Sharp Bldg., Lincoln, Neb., ALR
Petryshyn, Walter A., 140 E. 54th St., New York 22, N. Y., ALR
Plett, John V., 140 Pennsylvania Bldg., Butte, Mont., ALR
Pliskow, Harold, 5057 Woodward Ave., Detroit 2, Mich., OALR
Posey, William Campbell, Jr., 600 Suburban Square Bldg., Ardmore, Pa., OP
Preston, Ralph R., 704 Nat'l Reserve Bldg., Topeka, Kan., ALR
Preston, Richard A., 1521 N. Broadway, Santa Ana, Calif., OP
Pritchard, Dale B., Savings Bank Bldg., Ithaca, N. Y., OP
Puerner, Joseph W., 503 Third St., Niagra Falls, N. Y., ALR
Putney, Bernard Stephen, 170 Genesee St., Geneva, N. Y., ALR

Quick, Carl M., 3 N. Main St., Mansfield, Ohio, ALR

Radl, Cyril Joseph, Wood Block, Manitowoc, Wis., OP
Ralph, Fenn Tompkins, 50 E. 72nd St., New York 21, N. Y., OP
Ravdin, Emile Meyer, 4036 Wilshire Blvd., Los Angeles 5, Calif., OP
Reardon, William T., 207 W. 29th St., Wilmington 2, Del., ALR
Reed, John Hamilton, 402 Grand Theatre Bldg., Atlanta 3, Ga., OP
Reichert, Donald J., 24 W. Villard, Dickinson, N. D., OP
Reilly, William McKinley, Jr., 7 W. Wheeling St., Washington, Pa., OP
Reiter, Adolph F., 516 Locust St., McKeesport, Pa., ALR
Richards, William Wilson, U. S. Public Health Service Hospital, 15th Ave. and Lake St., San Francisco 18, Calif., OP
Ringer, Edward L., Landauer Bldg., Lincoln, Ill., ALR
Roberts, Henry James, Petroleum Bldg., Big Springs, Texas, OP
Robertson, Robert Brewer, 69 Maple Ave., Red Bank, N. J., ALR
Rogers, Layton S., 226 N. Greenleaf Ave., Whittier, Calif., ALR
Rose, John Edwin, 309 Medical Arts Bldg., Winnipeg, Man., Canada, OP
Rosehill, David Bernard, 48 E. Santa Clara St., San Jose 13, Calif., OP
Rosenberg, Alan J., 450 Sutter St., San Francisco 8, Calif., OP
Rosenvold, Lloyd Kenneth, 700 Main St., Montrose, Colo., ALR
Rudo, Alvin D., Latrobe Bldg., 2 E. Read St., Baltimore 2, Md., ALR
Rufe, John Robert, 2003 Bryant Bldg., Kansas City 6, Mo., OP

Sale, Charles Steven, 1010 Murchison Bldg., Wilmington, N. C., ALR
Sage, Harry M., Jr., 9 Buttles Ave., Columbus 8, Ohio, OP
Schaefer, Arthur John, 389 Linwood Ave., Buffalo 9, N. Y., OP
Schafer, William Charles, 1312 Bedford Rd., Washington, Ind., ALR
Schall, Roy F., 5058 Jenkins Arcade, Pittsburgh 22, Pa., ALR
Schlaegel, Theodore Frank, Jr., 624 Hume Mansur Bldg., Indianapolis 4, Ind., OP
Schmidt, Robert Thomas, 306 Bellin Bldg., Green Bay, Wis., ALR
Scruggs, James Harry, Jr., 2320 Columbus Ave., Waco, Texas, OP
Seale, Earl S., 126 Bay State Rd., Boston 15, Mass., OP
Seligman, Ewing, 405 N. Bedford Dr., Beverly Hills, Calif., ALR
Sellers, John Greaton, 413 Medical Arts Bldg., Norfolk 10, Va., ALR
Shainhouse, Arthur, 1217 48th St., Brooklyn 19, N. Y., OP
Shaw, Howard Arthur, 1231 Medical Arts Bldg., Minneapolis 2, Minn., OP
Shrader, Doyle A., 410 Schweiter Bldg., Wichita 2, Kan., ALR
Simonton, John Thayer, Blind Brook Lodge, Rye, N. Y., OP
Singer, Milton, 1920 N. W. Johnson St., Portland 9, Ore., OP
Sisson, George Allen, Medical Arts Bldg., Syracuse 2, N. Y., ALR
Skowron, John James, 4810 Milwaukee Ave., Chicago, Ill., OP
Smith, Burton Glancy, 2200 Santa Monica Blvd., Santa Monica, Calif., ALR
Smith, Edward Warren, 421 Huguenot St., New Rochelle, N. Y., OP
Smith, Marvin Dale, 522 Walnut St., Gadsden, Ala., OP
Smith, Robb Van Sittert, 261 E. Lincoln Highway, De Kalb, Ill., OP
Sneed, Ralph, 316 N. Congress, Jackson, Miss., ALR
Snyder, Stuart S., 525 E. 68th St., New York 21, N. Y., OP
Somers, Kenneth (Lt. Col.), Fitzsimons Army Hospital, Denver 8, Colo., ALR
Sovik, William Emil, 203 Mahoning Bank Bldg., Youngstown, Ohio, OP
Speas, Robert C., 402-403 Tribune Bldg., Terre Haute, Ind., ALR
Spence, George David, 216 Third St., N.E., Charlottesville, Va., OP
Sprunt, Charles Worth, 1200 18th St., N. W., Washington 6, D. C., OP
Steinberg, Harry, 1137 Second St., Santa Monica, Calif., ALR
Steinmetz, Arthur Frank, 1237 B St., Hayward, Calif., OP
Steinmetz, John Richard, 109 E. 14th St., San Leandro, Calif., OP
Stern, Charles, 442 Bay Ridge Parkway, Brooklyn 9, N. Y., OP
Stern, Henry, 350 Central Park West, New York 25, N. Y., OP
Stern, Richard U. (Major), U. S. Army Hospital, Fort Devens, Mass., OP
Sternberg, Milton, 826 E. 61st St., Chicago 37, Ill., OP
Stertzbach, Charles Walter, 710 Dollar Bank Bldg., Youngstown 3, Ohio, OP
Stevens, Norman D., 806 Central Ave., Woodmere, L. I., N. Y., OP
Stokes, Joseph Jackson, Medical Arts Bldg., 384 Peachtree St., N. E., Atlanta 3, Ga., OP

Stokes, Julius Howard, 125 W. Cheves St., Florence, S. C., OP

Stoner, Lee Rowland, 1100 E. Genesee St., Syracuse 10, N. Y., ALR

Strand, Sherman O'Neil, 1530 Arizona Ave., Santa Monica, Calif., ALR

Strobel, Robert J., 221 Fifth St., Bismarck, N. D., ALR

Suares, John Covington, Medical Arts Bldg., Greenville, Miss., OP

Sullivan, T. Ward, 210 Main St., Hackensack, N. J., OP

Swartz, Morris, 18 Broad St., Lynn, Mass., ALR

Swiontkowski, Stanley Dennis, 4341 S. Archer Ave., Chicago 32, Ill., OP

Swirsky, Albert, 1600 Randalia Rd., Fort Wayne, Ind., ALR

Tabb, William Granville, Jr., 511 Medical Arts Bldg., Atlanta 3, Ga., OP

Taylor, William Wickham, 319 Wainwright Bldg., Norfolk 10, Va., OP

Theobald, Pierce William, 307 N. Michigan Ave., Chicago 1, Ill., ALR

Thometz, Robert William, 501 W. Broadway, Missoula, Mont., ALR

Thompson, Roger T., 1507 Tower Ave., Superior, Wis., OP

Tindall, Robert Nelson, 16 Hampton Village Plaza, St. Louis 9, Mo., ALR

Tirico, Joseph G., 312-316 Spurgeon Bldg., Santa Ana, Calif., OP

Tokar, V. Raymond, 104 W. Clark St., Champaign, Ill., OP

Toll, William George, 56 E. 72nd St., New York 21, N. Y., OP

Traub, Paul Miller, 124 Dubois St., Newburgh, N. Y., ALR

Turnbull, Frederick Myles, Jr., 1930 Wilshire Blvd., Los Angeles 5, Calif., ALR

Tyner, George St. George, 324 Metropolitan Bldg., Denver 2, Colo., OP

Updegraff, Robert Rice, Veterans Administration Hospital, Iowa City, Iowa, ALR

Updegraff, William Rice, 607 Equitable Bldg., Des Moines 9, Iowa, ALR

Vaughan, James Baker, 129 Delaware Rd., Kenmore 17, N. Y., OP

Vegliante, Michael E., 174 Bradley St., New Haven, Conn., OP

Vetromile, Gerard A., 155 W. Merrick Rd., Freeport, L. I., N. Y., OP

Wagman, Ora Harold, 395 Commonwealth Ave., Boston 15, Mass., OP

Ward, James Patrick, 881 Lafayette St., Bridgeport 4, Conn., ALR

Wasmund, Clarence William, Interstate Clinic, Red Wing, Minn., OP

Watkins, John Gibson, Jr., 831 Donaghey Bldg., Little Rock, Ark., OP

Weiner, Harold, 122 Academy St., Poughkeepsie, N. Y., ALR

Weingeist, Samson, 30-74 36th St., New York (Queens), N. Y., OP

Weinstein, Irvin, Veterans Administration, 252 7th Ave., New York, N. Y., OP

Weisberg, Howard Kenneth, 1095 Park Ave., New York 28, N. Y., OP

Weixel, Francis X. (Major), 3700th Medical Group, Lackland Air Force Base, San Antonio, Texas, OP

Welch, John Gwyn, 620 N. Duke St., Lancaster, Pa., ALR

Welfeld, Alvan A., 1801 Eutaw Place, Baltimore 17, Md., ALR

Werner, George B., 42 Riverside Dr., Binghamton, N. Y., OP

West, Elmer J., Veterans Administration Hospital, Fort Thomas, Ky., ALR

West, Joseph Warren, 906 Olive St., St. Louis 1, Mo., ALR

Whiteley, Edward Jenner (Lt. Col.), U. S. Army Hospital, Fort Leavenworth, Kan., ALR

Whitten, Richard Hamilton (Capt.), U. S. Army Hospital, Camp Chaffee, Fort Smith, Ark., OP

Wholihan, John Wood, 102 Lydia St., Jackson, Mich., OP

Widener, George Hoy, 117 Guthrie Bldg., Paducah, Ky., OP

Wiesen, Arnold Maxwell, 39 Woodhull Ave., Riverhead, L. I., N. Y., ALR

Wildgen, Bernard C., 416 Hackley Union Nat'l Bank Bldg., Muskegon, Mich., OP

Wiley, George A., 1111 N. Lee St., Oklahoma City 3, Okla., OP

Willcockson, Thomas H., Yankton Clinic, 400 Park Ave., Yankton, S. D., OP

Wilson, George Clark, 4524 Sherman Oaks Ave., Sherman Oaks, Calif., ALR

Wood, William L., 47 Bay State Rd., Boston 15, Mass., ALR

Woodburn, Chester Charles, Jr., University Hospitals, Iowa City, Iowa, ALR

Woodley, Laurence Stephenson, 604 25th Ave., Tuscaloosa, Ala., ALR

Woolmington, Merrill H., Colonial Bldg., Wayne, Pa., OP

Worcester, John Thompson, 220 Engle St., Englewood, N. J., OP

Zeldin, Abraham Joel, 56 Ontario St., St. Catharines, Ont., Canada, ALR

The following candidates have qualified for Fellowship in the Academy except in some single detail—certification, membership in a local medical society, or the signatures of sponsors. These deficiencies are expected to be made up before the Annual Business Meeting.

Applewhite, Norman Elliot, 15 E. First St., Reno, Nev., ALR

Benedict, Walter Hanford, Mayo Clinic, Rochester, Minn.

Droege, Fred D., 208 Vanskike Bldg., Sarasota, Fla.

Harper, John Y., 849 Fifth St., Port Arthur, Texas

Johnson, Norman Edward, Manhattan Eye, Ear and Throat Hospital, 210 E. 64th St., New York 21, N. Y.

Jones, Ira Snow, 73 E. 71st St., New York 21, N. Y.

Knauff, Harry A., Wadsworth, Kan., ALR

Kreit, Warren William, 940 Jefferson Bldg., Peoria, Ill.

Leer, Ralph Harrison, Box 2597, Juneau, Alaska

O'Brien, Thomas Paul, 309 Wilson Bldg., Corpus Christi, Texas, OP

Peebles, Jonathan Blythe, Jr., U. S. Public Health Service Hospital, New Orleans, La., OP

Perley, Edward Peter, 524 E. 20th St., New York 9, N. Y.

Scillieri, John, 660 Broadway, Paterson 4, N. J.

MOTION PICTURE
THE EMBRYOLOGY OF THE EYE

Please note change in time of presentation.

The lecture and free demonstration by Dr. Harold F. Falls of the Academy film "The Embryology of the Eye" will be included in a program of films (see Special Scientific Programs, p. 707) to be presented on Thursday, October 16, at 8:00 p.m. It will not be shown at 4:30 p.m., Monday, October 13, as originally scheduled.

PROGRAM IN BRIEF

1952 Meeting — Chicago

SATURDAY, OCTOBER 11, 1952

9:00 a.m.—Council Meeting — Private Dining Room 4

8:00 p.m.—Special Scientific Program: Otosclerosis Study Group—Private Dining Room 14

SUNDAY, OCTOBER 12, 1952

9:00 a.m.—Registration—Foyer to Grand Ball Room

9:00 a.m.—Ladies' Registration—Private Dining Rooms 15 and 16

9:00 a.m.—Official Opening of Exhibits: Scientific—Red Lacquer Room
Technical—Exhibition Hall and Red Lacquer Room

10:00 a.m.—Meeting of Joint Committee on Industrial Ophthalmology—Dr. Kuhn's Suite

10:00 a.m.—Teachers' Section: Ophthalmology—Crystal Room

10:00 a.m.—Teachers' Section: Otolaryngology—Private Dining Room 14

12:30 p.m.—Home Study Courses Faculty Luncheon—Private Dining Room 9

4:30 p.m.—President's Reception — Grand Ball Room

8:00 p.m.—Special Scientific Program: Committee on Conservation of Hearing and Otosclerosis Study Group—Grand Ball Room

8:00 p.m.—Special Scientific Program: Joint Committee on Industrial Ophthalmology — Private Dining Room 14

MONDAY, OCTOBER 13, 1952

9:00 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

9:30 a.m.—Joint Scientific Session — Grand Ball Room

10:15 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

11:30 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Rooms 14 and 17

3:15 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Rooms 14 and 17

4:30 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

6:30 p.m.—Alumni Dinners

TUESDAY, OCTOBER 14, 1952

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ball Room

9:00 a.m.—ENT Instruction Courses—Seventh Floor

9:00 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

10:15 a.m.—ENT Instruction Courses—Seventh Floor

10:15 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

11:30 a.m.—ENT Instruction Courses—Seventh Floor

11:30 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

12:30 p.m.—Technical Exhibitors Luncheon—Private Dining Room 9

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

3:15 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

4:30 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

5:30 p.m.—Dinner Meeting of American Association of Eye, Ear, Nose and Throat Society Secretaries—Private Dining Room 18

8:00 p.m.—Special Scientific Program: American Orthoptic Council and American Association of Orthoptic Technicians—Private Dining Room 14

8:00 p.m.—Special Scientific Program: Committee on Plastic Surgery—Grand Ball Room

WEDNESDAY, OCTOBER 15, 1952

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ball Room

9:00 a.m.—ENT Instruction Courses—Seventh Floor

10:15 a.m.—ENT Instruction Courses—Seventh Floor

11:30 a.m.—ENT Instruction Courses—Seventh Floor

12:45 p.m.—Council Meeting Luncheon—Private Dining Room 5

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

3:15 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

4:30 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

7:00 p.m.—Annual Dinner—Grand Ball Room

THURSDAY, OCTOBER 16, 1952

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ball Room

9:00 a.m.—ENT Instruction Courses—Seventh Floor

10:15 a.m.—ENT Instruction Courses—Seventh Floor

11:30 a.m.—ENT Instruction Courses—Seventh Floor

12:45 p.m.—Faculty Luncheon—Private Dining Room 14

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ball Room

2:00 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

3:15 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

4:30 p.m.—EYE Instruction Courses—Seventh Floor and Private Dining Room 17

5:30 p.m.—Business Meeting—Crystal Room

8:00 p.m.—Motion Pictures — Grand Ball Room

FRIDAY, OCTOBER 17, 1952

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ball Room

9:00 a.m.—Joint Scientific Session with American Society of Ophthalmologic and Otolaryngologic Allergy — Private Dining Room 14

2:00 p.m.—Joint Scientific Session with American Society of Ophthalmologic and Otolaryngologic Allergy—Grand Ball Room

SCIENTIFIC SECTIONS

JOINT SESSION

Monday, October 13, 1952
9:00 a.m.
GRAND BALL ROOM

Presiding Officer:
JAMES MILTON ROBB, M.D.
President

Secretary:
KENNETH L. ROPER, M.D.

Call to Order.....The President

Announcements
George E. Shambaugh, Jr., M.D.
General Chairman

Introduction of Guest of Honor
Fletcher D. Woodward, M.D.
Charlottesville, Va.

Address by the President
"As is your pathology so is your practice"

Address by the Guest of Honor
The Widening Aspects of Otolaryngology

Presentation of Honor Keys

SCIENTIFIC PROGRAM

The Pathology of Some Systemic Diseases Routinely Diagnosed by the Ophthalmologist and Otolaryngologist

Plinn Morse, M.D.
Detroit, Mich.
BY INVITATION

Panel on the Atlases of Pathology
Introduction
William L. Benedict, M.D.
Rochester, Minn.

Early History
Harris P. Mosher, M.D.
Boston, Mass.

The Army Medical Museum and the Registry of Pathology
Brigadier General
George R. Callender
Washington, D. C.
BY INVITATION

Development of Tumor Registries
Brigadier General
Elbert DeCoursey
Washington, D. C.
BY INVITATION

Development of the Atlases
Colonel J. E. Ash (Retired)
Washington, D. C.

Objectives of the Pathology Committees
Brittain F. Payne, M.D.
New York, N. Y.

Selection of Pictures and Technics—Ophthalmology
Helenor Campbell Wilder
Washington, D. C.

Selection of Pictures and Technics—Otolaryngology
Muriel Raum, M.D.
Washington, D. C.
BY INVITATION

Text and Atlas of Ophthalmic Pathology
Jonas S. Friedenwald, M.D.
Baltimore, Md.

Value of Histopathology in a General Medical Curriculum
Edmund P. Fowler, Jr., M.D.
New York, N. Y.

AUTHORS OF PAPERS

All slides or films must be in the hands of the projection operator before the opening of each session.

Please be prepared to submit papers to the recorder immediately following presentation.

SECTION ON OTOLARYNGOLOGY

Monday, October 13, 1952
2:00 p.m.
GRAND BALL ROOM

Presiding Officer:
JAMES MILTON ROBB, M.D.
President

Secretary:
HOWARD P. HOUSE, M.D.

MEGA-ESOPHAGUS (DYSTONIA) AND ESOPHAGITIS

Owen H. Wangensteen, M.D.
Minneapolis, Minn.
BY INVITATION

Discusser:
Paul H. Holinger, M.D.
Chicago, Ill.

During the past few years, some light has been shed upon the nature of some of the bizarre and little-understood esophageal lesions. It would appear that mega-esophagus is primarily dystonia of the esophagus. There is hypertonus of the distal segment and atony of the more proximal reaches of the esophagus. In fact, the condition behaves very much like Hirschsprung's disease in the colon and rectum. And similarly, parasympathetic ganglion cells are absent in Auerbach's plexus, much as in Hirschsprung's disease of the colon.

During recent years, considerable information has accumulated to indicate a great sensitivity of the esophagus to injury by acid peptic juice. All operative procedures directed at the cure of mega-esophagus in which the physiologic sphincter of the terminal esophagus is bypassed invite esophageal erosion and hemorrhage. In other words, the Heyrovsky-Grondahl operation of esophagogastrostomy is out of order in that it disposes to esophagitis. Similarly, the Wendel procedure of open cardioplasty is also an unacceptable procedure. The Heller operation of extramucosal myotomy does not appear to invite esophagitis. The best operation for the baglike, redundant, giant esophagus is straightening of the esophagus together with excision of the redundant terminal portion, accompanied by simultaneous excision of the entire acid-secreting area of the stomach. The straightened esophagus is anastomosed directly to the antrum; an extramucosal myotomy is done on the pyloric sphincter to insure satisfactory emptying of the vagotomized antrum.

A bloodless Heller myotomy is achieved in the hands of experienced endoscopists in a fairly large number of cases by hydrostatic dilatation. When, however, an attempt or two at dilatation of the hypertonic distal segment by this maneuver does not accomplish the objective, the patient should be subjected to operation. Dilatation of the esophagus by bougies is only a form of palliative treatment; it is not curative.

The unpredicted success of gastric resection undertaken for bleeding duodenal ulcers in ridding patients of esophagitis and/or stricture has definitely established the validity of the thesis that idiopathic strictures of the esophagus can be cured by gastric resection, much in the manner that a satisfactory gastric resection cures duodenal ulcer. Moreover, experiments in animals suggest quite definitely that the esophagus is more sensitive to injury by acid peptic juice than is any tissue lying near the stomach. Furthermore, experiments suggest rather definitely, too, that so-called "spontaneous perforation" of the esophagus is in all likelihood perforation of the esophagus due to acid peptic juice. It is also evident that the esophagus can be injured by bile and pancreatic juice. The esophageal mucosa is not injured by succus entericus. This latter circumstance suggests that the best manner in which to link up the alimentary tract following total gastrectomy is to anastomose the esophagus to the jejunum by the Roux Y principle of operation.

2:35 p.m.

COMMENTS ON THE PATHOLOGY OF THE PHARYNGEAL RECESS, EUSTACHIAN TUBES, TONSILS AND ADENOIDS

Herman Semenov, M.D.
Beverly Hills, Calif.

Discussers:
Henry L. Williams, M.D.
Rochester, Minn.
Ben H. Senturia, M.D.
St. Louis, Mo.

The problem of chronic pharyngitis, particularly follicular pharygnitis, is an old one and, needless to add, an impor-

tant one because it is so common. Much of the pathology is hidden from sight and lies within the nasopharynx. In treating the entire lymphatic apparatus of the throat we must constantly consider the quagmire of pathology found in the pharyngeal recess, tonsils and adenoids.

3:15 p.m.

BONE GRAFTING IN SADDLE NOSE
(A Motion Picture)

Otto W. Wickstrom, Capt. (MC) USN
Oakland, Calif.
and
Joseph R. Connelly, Comdr. (MC) USN
Philadelphia, Pa.

BY INVITATION

The saddle nose deformity is one of the common defects found in patients complaining of impaired nasal respiratory function. To correct such a defect several sources of material are available. Autogenous and preserved cartilage are used extensively. Disadvantages of cartilage are (a) tendency for the upper end of the graft to curl and (b) a softening of the strut over a period of time. Bone grafts have been found to maintain their contour and to provide the necessary support to the nasal dorsum. An easy and ample source of bone may be obtained from the iliac crest.

A motion picture illustrates the technic of this procedure.

3:40 p.m.

A NEW METHOD OF OBTAINING EUSTACHIAN TUBE CLOSURE AT TYMPANOMASTOIDECTOMY

Louis E. Adin, Jr., M.D.
Dallas, Texas
and
Donald A. Corgill, M.D.
McKinney, Texas

Discussers:

Philip E. Meltzer, M.D.
Boston, Mass.
Percy E. Ireland, M.D.
Toronto, Ont., Canada

Since the advent of mastoid surgery the problem of intermittent or continued discharge from the operated ear has concerned otologists and patients alike. Most otologists agree that a patent eustachian tube is by far the most common cause. A historical survey of the methods which have been tried to obtain closure of the eustachian tube will be given. A short motion picture will be shown to demonstrate how autogenous cancellous bone is obtained for use as a free transplant into the cleansed eustachian tube at tympanomastoidectomy for the purpose of securing permanent closure. The results in a series of cases operated upon by this method will be presented.

4:20 p.m.

THE EFFECT OF PROGRESSIVELY BUFFERED SOLUTION OF EPHEDRINE ON THE RESPIRATORY NASAL MUCOUS MEMBRANE

Noah D. Fabricant, M.D.
Chicago, Ill.

Discusser:

Leland G. Hunnicutt, M.D.
Pasadena, Calif.

This paper presents a summary of the histologic findings produced by a progressively buffered solution of ephedrine (bearing pH values 2.5; 3.0; 3.5; 4.0; 4.5; 5.0; 5.5; 6.0; 6.5; 7.0; 7.5; 8.0; 8.5; 9.0; 9.5; 10.0; 10.5; 11.0; 11.5 and 12.0) on the nasal mucous membrane of a large group of laboratory animals receiving intranasal instillations three times daily for a period of 30 days. An attempt is made to correlate tissue changes to pH values, and to discuss some of the implications of nasal pH in terms of nasal physiology, nasal medication, and clinical practice.

SECTION ON OPHTHALMOLOGY

Tuesday, October 14, 1952
9:00 a.m.

GRAND BALL ROOM

Presiding Officer:
GEORGIANA D. THEOBALD, M.D.
First Vice-President

Secretary:
KENNETH L. ROPER, M.D.

PATHOLOGIC FINDINGS IN AN EYE WITH A NONHEALING CATARACT INCISION
(A Clinicopathologic Case Report)

Wilfred E. Fry, M.D.
Philadelphia, Pa.

The patient, age 69, was a healthy adult female who had mild diabetes easily controlled on an adequate diet. On April 20, 1950, she had an intracapsular cataract extraction with a peripheral iridectomy in

the right eye with no complications and 6/6+ vision. A year and three months later she had an intracapsular extraction of the second cataract by a similar technic, namely peripheral iridectomy and intracapsular extraction, with no complications. The incision was closed with mild chromic catgut sutures. There were apparently no complications until eight days later when following sharp pain there was minimal iris incarceration at the temporal pillar. A prolapse of iris developed. The wound was repaired on four occasions. None of the repairs were successful and the incision would not heal. The eye was enucleated on August 27, 1951.

The pathologic findings include the nonhealing corneal incision and a subacute subchoroidal hemorrhage. The latter may be responsible for the nonhealing cataract incision.

9:10 a.m.

PRESENTATION OF NEW METHODS AND NEW INSTRUMENTS

9:20 a.m.

CONGENITAL OCULAR APRAXIA

(Jackson Memorial Lecture)

David G. Cogan, M.D.
Boston, Mass.

10:00 a.m.

THE KRONLEIN OPERATION

(A Motion Picture)

Algernon B. Reese, M.D.
New York, N. Y.

The anatomy concerned in performing the operation is demonstrated on the cadaver and then the technic of the operation is shown. The features emphasized are (1) the Berke incision, (2) the intact periosteum, (3) the oscillating saw.

In color with sound.

10:20 a.m.

OPHTHALMOPLEGIC MIGRAINE: A DISCUSSION OF ITS PATHOGENESIS WITH A REPORT OF THE PATHOLOGIC FINDINGS IN A CASE OF RECURRENT OCULOMOTOR PALSY

David O. Harrington, M.D.
and
Milton Flocks, M.D.
BY INVITATION
San Francisco, Calif.

Discussers:

Frank B. Walsh, M.D.
Baltimore, Md.
Hugh C. Donahue, M.D.
Boston, Mass.

The clinical syndrome known as ophthalmoplegic migraine has excited wide interest, in spite of its rarity, since it was first defined by Charcot in 1890. Its pathogenesis is unknown. Its relationship to migraine or hemicrania simplex is seriously questioned by Burki and Duke-Elder, who feel that "the syndrome is the clinical manifestation of several distinct pathologic states." The five cases clinically diagnosed as ophthalmoplegic migraine which have come to autopsy have shed little light on the problem; in fact, have only added to the confusion.

In the present article a known mechanism in the production of recurrent oculomotor palsy (hippocampal gyrus herniation) is described and discussed. A case of clinically typical ophthalmoplegic migraine is reported in which, at autopsy, there was demonstrated a herniation of the uncus or hippocampal gyrus over the edge of the tentorium with pressure on the third nerve.

The pathogenesis of migraine (hemicrania simplex) is discussed. An attempt is made to reconcile the theories of its etiology with a new theory of the pathogenesis of ophthalmoplegic migraine.

11:05 a.m.

HEMANGIOMA OF CONJUNCTIVA: REMOVAL WITH PEDICLE GRAFT FOR REPAIR

(A Motion Picture)

Wendell L. Hughes, M.D.
Hempstead, N. Y.

The film detects a hemangiomatous lesion of the conjunctiva over the medial rectus in the medial canthus. A method of removal, the multiple structure of the tumor, the pulsation of the nutrient vessels and the method of removal are demonstrated. The repair was carried out by a preliminary covering of the medial rectus with a thin layer of fascia bulbi and then a conjunctival flap was turned down from the upper fornix and sewn in place.

11:25 a.m.

THE EFFECT OF CORTISONE AND HYDROCORTISONE IN CORNEAL ULCERS: A CLINICAL AND EXPERIMENTAL STUDY

Phillips Thygeson, M.D.
San Jose, Calif.
and
Michael J. Hogan, M.D.
San Francisco, Calif.

Discussers:

James H. Allen, M.D.
New Orleans, La.
Irving H. Leopold, M.D.
Philadelphia, Pa.

Cortisone and hydrocortisone proved to be remarkably effective in the treatment of marginal corneal ulcers and infiltrates associated with catarrhal conjunctivitis and blepharoconjunctivitis. Since no organisms are found in this type of ulcer, it is believed that it is toxic or allergic in origin and that the cortisone effect is antiallergic and not antibacterial. This was confirmed by the finding that the hormones had no significant effect on the primary conjunctival and lid disease. Cortisone was used in a limited number of cases of hypopyon ulceration of varied etiology and was found to have no significant effect.

In experimental corneal ulceration in guinea pigs produced by intracorneal injection of Staphylococcus aureus, Pseudomonas aeruginosa, beta hemolytic streptococcus, and Diplococcus pneumoniae, type II, cortisone administered topically and systemically was totally ineffective in modifying the experimental disease and did not interfere with the protective effect of antibiotics used simultaneously.

The literature dealing with the effects of ACTH and cortisone in bacterial and viral infections is summarized and discussed.

12:10 a.m.

LAMELLAR RESECTION OF THE SCLERA FOR RETINAL DETACHMENT

(A Motion Picture)

Milton L. Berliner, M.D.
New York, N. Y.

The purpose of this film is to draw attention to the procedure of lamellar resection of the sclera which consists of the removal of tangential strips of three fourths of the scleral thickness in conjunction with diathermy coagulation. The sclera is shortened by means of buried silk sutures which causes an infolding of the surgically thinned area. The operation is simple to perform and does not expose the globe to any of the dire complications attending penetrating scleral resections.

SECTION ON OTOLARYNGOLOGY

Tuesday, October 14, 1952
2:00 p.m.
GRAND BALL ROOM

Presiding Officer:
PERCY E. IRELAND, M.D.
Second Vice-President
Secretary:
HOWARD P. HOUSE, M.D.

PRESENTATION OF NEW METHODS AND NEW INSTRUMENTS

2:10 p.m.

SURGICAL REMOVAL OF PAROTID GLAND TUMORS

James H. Maxwell, M.D.
and
Robert W. Buxton, M.D.
BY INVITATION
A. James French, M.D.
BY INVITATION
Ann Arbor, Mich.

Discussers:

A. James French, M.D.
Ann Arbor, Mich.
BY INVITATION
Frank Lathrop, M.D.
Boston, Mass.

This paper is based upon a complete re-evaluation and reclassification of tumors of the parotid gland removed in the University of Michigan Hospital in the past eighteen years. An attempt has been made, through correlation of detailed histopathologic examinations and complete follow-up studies of patients, to determine how extensive the primary excision should be and to decide in which types of tumors a prophylactic neck dissection should be considered.

2:50 p.m.

SYMPOSIUM: THE SURGICAL MANAGEMENT OF AURAL CHOLESTEATOMA

Why I Remove the Matrix

Theodore E. Walsh, M.D.
St. Louis, Mo.

The theory of pressure necrosis from the expansion of the cholesteatoma due

to accumulation of desquamated epithelial cells has largely been disproved by clinical observation. Cases have been observed with no obstruction to the removal of desquamated epithelial cells which at operation have been found to have a fistula into the horizontal canal or other parts of the labyrinth.

Sections of ossicles removed from cases of cholesteatoma have demonstrated an ingrowth of the matrix through the periosteal bony layer into the endosteal layer with a decalcification of the calcified cartilaginous rests and subsequent necrosis. Similar observations have been made in temporal bones removed at autopsy in cases of cholesteatoma. The ability of the matrix to invade blood spaces and its ability to cause decalcification in the calcified cartilaginous rests makes its imperative that this matrix be removed.

Histologic evidence is presented together with case histories supporting this view.

Why and When I Do Not Remove the Matrix

Shirley H. Baron, M.D.
San Francisco, Calif.

Conservation of hearing is the basic motive for preserving the matrix.

A number of otologists feel that the matrix, per se, has innate, constant biologic activity and have urged its complete eradication at any cost, even at the expense of the hearing.

Actually, as shown by clinical experience, the squamous epithelium of the matrix, when restored to a healthy, dry state, is innocuous and useful. Progression of bony necrosis underlying the preserved matrix is the rare exception rather than the rule.

Preservation of the matrix leads to less radical mastoid surgery and better postoperative hearing levels.

Methods, indications and contraindications for preserving the matrix will be presented.

Histopathology, Pathogenesis and Summary

John R. Lindsay, M.D.
Chicago, Ill.

The two common types of cholesteatoma found in the middle ear spaces have been that which follows destruction of the drum membrane and middle ear mucosa by a necrotizing otitis and that which develops more or less insidiously in the epitympanum.

The incidence of the former has been decreased by antibiotic therapy.

The latter type appears not to be decreased and comprises the majority of cases coming for treatment at the present time.

Discussion of histopathology and pathogenesis will be directed mainly to this latter group.

The discussion of the previous speakers on management of the matrix will be summarized.

3:40 p.m.

AN OTOLOGICAL SEMINAR
(A Motion Picture)

H. G. Kobrak, M.D., Ph.D.
Chicago, Ill.

The film shows the physical aspects of a number of ear diseases. The concept of impedance is explained and the ear diseases which are demonstrated in the film are analyzed as mass lesions, stiffness lesions and friction lesions. "Normal Eardrum" and "Normal Nasopharynx" are shown first. The following diseases are demonstrated:

1. Serous Otitis
   a. In children with adenoid tissue in nasopharynx.
   b. In adults with tumor in nasopharynx. Typical audiograms are given. High tone losses in conduction lesions are explained.
2. A glomus tumor of the middle ear is shown as an example of a mass lesion. Audiogram, biopsy, clinical course.
3. Osteogenesis imperfecta with blue sclera and Schwarze's sign on the promontory is shown as a stiffness lesion. Histology of otosclerosis, sections taken through phase microscope and by ordinary microscope.
4. The audiograms of patients with inner ear lesions demonstrating recruitment are shown. The concept of recruitment is demonstrated by audiovisual methods as it appears to the patient.
5. Acoustic trauma (high speed camera work) and stroboscopic exposures are shown in animal experiments.

4:00 p.m.

PHYSIOLOGY OF THE SPHINCTER MECHANISM OF THE LARYNX

Joel J. Pressman, M.D.
Beverly Hills, Calif.

Discussers:

A. C. Furstenberg, M.D.
Ann Arbor, Mich.
Thomas C. Galloway, M.D.
Evanston, Ill.

The sphincteric action of the larynx is becoming increasingly important from the standpoint of the application of therapeutic surgical principles. An intimate knowledge of its physiology is therefore fundamental, and this will be described in so far as it is of practical importance.

4:40 p.m.

EMERGENCY TREATMENT OF CARDIAC ARREST IN THE OPERATING ROOM
(A Motion Picture)

Robert M. Hosler, M.D.
Cleveland, Ohio

BY INVITATION

Cardiac arrest in the operating room is the enigma and challenge of present-day surgery and anesthesia. It can occur in cases handled by the beginning intern as well as in those of the skilled surgeon. It can happen during a simple operation as well as during a complicated one. It can take place under local or regional anesthesia as well as during general anesthesia.

Obviously the best therapy for this catastrophe is its prevention. Often there are obscure danger signals which herald impending disaster. To ignore these and concentrate on cardiac resuscitation alone would be like locking the barn after the horse had been stolen. Nevertheless, when confronted with this extraordinary catastrophe, if one is to be successful in meeting this challenge, a definite preconceived pattern or plan of attack must be understood. One must reflexly know the things to do and the things not to do during those critical moments while life hangs in the balance. One does not have time to improvise methods, as a time limitation of three to five minutes must be overcome, otherwise the brain has suffered irreversible damage.

The resuscitation procedure is divided into two separate and distinct steps; (1) the re-establishment of the oxygen system, and (2) the restoration of the heart beat. An important subdivision of the first, is the immediate and skillful introduction of an intratracheal tube. The attention should be focused on the first step, as the restoration of the heart beat is secondary. The two conditions of cardiac stoppage encountered in the operating room are cardiac standstill and ventricular fibrillation. Each must be dealt with differently.

A colored motion picture will demonstrate the procedure.

Most failures can be attributed to the following causes: (1) lack of a plan of attack, (2) failure to overcome the time limitation, (3) inadequate oxygenation, (4) inadequate absorption of carbon dioxide, (5) inherent cardiac disease.

A two-day practical course in this subject is given once a month in Cleveland, Ohio, by Drs. Beck and Hosler. This course is sponsored by the Cleveland Heart Society.

SECTION ON OPHTHALMOLOGY

Wednesday, October 15, 1952
9:00 a.m.

GRAND BALL ROOM

Presiding Officer:
JOHN S. MCGAVIC, M.D.
Third Vice-President

Secretary:
KENNETH L. ROPER, M.D.

A TRIBUTE TO DR. WALTER B. LANCASTER

James J. Regan, M.D.
Boston, Mass.

9:10 a.m.

STRABISMUS SYMPOSIUM

Introduction

Kenneth C. Swan, M.D., Chairman
Portland, Ore.

Adaptive Mechanisms

Hermann C. Burian, M.D.
Iowa City, Iowa

Horizontal Deviations

Francis H. Adler, M.D.
Philadelphia, Pa.

Vertical Deviations
Harold W. Brown, M.D.
New York, N. Y.

Principles of Treatment
Frank D. Costenbader, M. D.
Washington, D. C.

Surgery
C. Allen Dickey, M.D.
San Francisco, Calif.

Conclusions
Kenneth C. Swan, M.D.
Portland, Ore.

SECTION ON OTOLARYNGOLOGY

Wednesday, October 15, 1952
2:00 p.m.

GRAND BALL ROOM

Presiding Officer:
JAMES MILTON ROBB, M.D.
President
Secretary:
HOWARD P. HOUSE, M.D.

FIRE AND EXPLOSION HAZARDS WITH FLAMMABLE ANESTHETIC AGENTS, AND THEIR CONTROL

George J. Thomas, M.D.
Paul G. Guest, Electrical Engineer
BY INVITATION
Pittsburgh, Pa.

The lecture and demonstration will cover the following:

1. Factors conducive to fire and explosion with flammable anesthetic agents.
2. The value of conductive floors, efficient grounding, and wet towels in minimizing explosion hazards in anesthetizing locations.
3. A demonstration of electrostatic charges igniting flammable anesthetic mixtures.
4. The danger of various types of material in producing electrostatic charges of sufficient intensity to ignite flammable anesthetic agents.
5. A demonstration of the danger of nonconductive mattresses on operating and delivery room tables.
6. The value of nonflammable anesthetic agents in the prevention of fire and explosion in hospitals.

2:30 p.m.

SYMPOSIUM: THE MODERN TREATMENT OF MAXILLARY SINUSITIS

Nonsurgical Treatment of Acute and Chronic Maxillary Sinusitis
O. E. Van Alyea, M.D.
Chicago, Ill.

Cases of acute maxillary sinusitis tend to heal spontaneously; those which persist do so because of drainage impairment. In the early stages efforts are directed toward relief of the symptoms and prevention of complications, but as the acute nasal engorgement recedes, measures both local and general are introduced which aid in the early termination of the infection.

In the management of chronic sinusitis simple measures if properly applied suffice in the majority of cases. Periodic irrigation is carried out and progress of the case is noted by occasional roentgen studies with iodized oil and by examination of the exudate removed at time of lavage.

Considerable improvement often follows a simple infraction of a middle turbinate or the removal of a few polyps. In other cases more extensive procedures such as septal resection, control of allergy, correction of systemic disorders or treatment of an allied sinusitis may be indicated.

Diagnosis of Chronic Maxillary Sinusitis
Frederick T. Hill, M.D.
Waterville, Me.

While the development of chemotherapy and antibiotic therapy, together with a better understanding of nasal physiology and of the etiologic factors involved, has resulted in less frequent employment of surgical measures in cases of chronic maxillary sinusitis, the necessity for drainage or the presence of irreversible pathologic states may require surgical intervention. The indications for any such procedure depend upon careful study of each individual case. While the possibility of complications may make necessary at times the use of radical procedures, every effort should be made to preserve the normal functions of the parts involved. The operation should be fitted to the patient, not the patient to the operation.

Technic in Surgical Therapy: End Results

Lawrence R. Boies, M.D.
Minneapolis, Minn.

The ideal "antrum window" (intranasal antrostomy) is large in the inferior meatus and is made with a minimum amount of trauma. Adequate permanent drainage is accomplished. Most of the interior of the antrum can be visualized except the anterior wall. In the majority of cases a sufficient amount of diseased membrane can be removed.

The "radical antrum" (Caldwell-Luc) can be reserved almost entirely for cases in which the possibility of malignancy is suspected. A technic is described in which there is practically no instrumentation within the nasal fossa.

When packing can be avoided there is less reaction. Limitation of local after-treatment to dry suction is desirable.

In this day, a cure is practically certain with either method of surgical treatment.

3:10 p.m.

STRUCTURAL CHANGES IN THE CILIATED EPITHELIAL CELLS DURING ACUTE UPPER RESPIRATORY INFECTIONS

(With kodachrome illustrations)

William T. K. Bryan, M.D.
and
Marian Pfingsten Bryan
BY INVITATION
St. Louis, Mo.

Discusser:
George E. Shambaugh, Jr., M.D.
Chicago, Ill.

Nasal cytology has been studied by adapting the staining technic of Papanicolaou to nasal secretions. Observations on series of consecutive smears taken twice a day throughout the course of acute upper respiratory infections are reported. Duplicate slides in Wright's stain were made for comparison.

The Papanicolaou stain, which has proved so valuable for the recognition and study of malignant cells, preserves and brings out details not previously seen in nasal cytology. It makes possible the accurate differentiation of the types and conditions of the epithelial cells exfoliated in nasal secretions. Intracellular details and cilia are thus clearly demonstrated in clinical nasal smears with this technic.

The ciliated cells in acute upper respiratory infections undergo a definite pattern of degeneration which is suggestive of virus activity and which may be used in clinical diagnosis of the "common cold." These changes will be illustrated with kodachrome slides taken of individual cells at high magnification.

The importance of studying the cytology of all cells in nasal exudates in series with various stains is emphasized. Probably no one stain is sufficient to see all cellular details. New patterns added to those previously studied, when carefully correlated with clinical symptoms, can increase our understanding of nasal pathology.

3:40 p.m.

SELECTED PROBLEMS IN THE DIAGNOSIS OF LARYNGEAL CARCINOMA

Stanton A. Friedberg, M.D.
Linden J. Wallner, M.D.
Chicago, Ill.

Discussers:
Henry B. Orton, M.D.
Newark, N. J.
Alden H. Miller, M.D.
Los Angeles, Calif.

The aim of this report is to present some challenging aspects in the diagnosis and evaluation of laryngeal carcinoma as illustrated by photographic studies. The subject matter is divided into six categories: (1) leukoplakia and vocal cord carcinoma; (2) the relation between the appearance and actual extent of carcinoma (a comparison of views of the lesion and the operative specimen); (3) the dearth of symptoms in extracordal carcinoma; (4) variations in diagnostic aids; (5) some problems in the choice of therapy; and (6) carcinoma of the larynx and tuberculosis. A group of 85 patients with carcinoma of the larynx is represented. The photographic material comprises approximately 60 kodachrome views of lesions and specimens from which may be chosen illustrative examples in each of the above subdivisions.

In the consideration of leukoplakia, attention is directed to recurrent forms simulating malignancy and to the gross similarity between keratosis of the larynx and carcinoma. Leukoplakia as a precursor of carcinoma is presented in graded sequence. Benign laryngeal lesions with leukoplakic surface alterations are described and illustrated.

The mirror or direct views of laryngeal cancer may or may not yield an accurate estimate as to the true extent of the pathology. This is borne out by comparisons between the appearance of the lesion and the surgical specimen in instances of cordal, endolaryngeal, extrinsic and subglottic growths.

Examples of extensive extracordal lesions in patients with practically no subjective symptoms bear out the relatively silent nature of certain forms of laryngeal cancer.

Planigrams, suspension laryngoscopy and roentgenographic studies with opaque media are shown to be of accessory value in delineating tumor size.

Certain aspects of treatment are considered in so far as they deal with decisions which were made regarding roentgen therapy, thyrotomy and laryngectomy in representative types of cases.

Finally, the gross similarity between tuberculosis and carcinoma of the larynx is portrayed by comparable lesions involving the epiglottis, arytenoids, ventricular bands and vocal cords.

4:20 p.m.

CAROTID ARTERY ANASTOMOSIS

John J. Conley, M.D.
New York, N. Y.

Discussers:

Lewis F. Morrison, M.D.
San Francisco, Calif.

Julius A. Weber, M.D.
Seattle, Wash.

Compulsory excision and ligation of the common and internal carotid arteries in the management of tumors of the head and neck has carried a mortality of approximately 50 per cent. In an effort to reduce the seriousness of this procedure two technics were developed. The first technic consists of the ipsilateral anastomosis of the cut end of the internal carotid artery to the cut end of the external carotid artery in the upper part of the neck. The second technic consists of a free autogenous vein graft anastomosed between the common and internal carotid arteries. The first technic or ipsilateral anastomosis, prevents in a relative manner the ischemia to the brain as a result of internal carotid artery ligation. The second technic, or autogenous vein graft anastomosis, maintains the intra-arterial pressure and prevents cerebral ischemia entirely.

The indications for a compulsory ligation or excision of the common and internal carotid arteries in radical surgery of the neck are associated with rupture of the internal or common carotid arteries, threatened rupture of the internal or common carotid arteries, malignant tumor directly invading the internal or common carotid arteries, and carotid body tumor. The same situation exists in traumatic rupture of the vessel or in arteriovenous aneurysm. This variety of indications does not permit the application of one technic in every instance, neither does it apply to the individuals who ordinarily would not die as a result of ligation of the common or internal carotid arteries.

It is hoped that as a result of intra-arterial pressure readings and other clinical investigations, these technics may be able to be applied to those individuals who would otherwise die as a result of ligation of the internal or common carotid arteries.

SECTION ON OPHTHALMOLOGY

Thursday, October 16, 1952
9:00 a.m.

GRAND BALL ROOM

Presiding Officer:
GEORGIANA D. THEOBALD, M.D.
First Vice-President
Secretary:
KENNETH L. ROPER, M.D.

MULTIPLE MALIGNANT MELANOMAS IN ONE EYE
(A Clinicopathologic Case Report)

David M. Rosen, M.D.
New York, N. Y.
and
Gardner Moulton, M.D.
Baltimore, Md.

BY INVITATION

Patient is a 52 year old white male who had been having visual disturbances in his left eye for four years. On examination there was found to be a large mass extending from the disc to the far temporal periphery. The globe was enucleated and submitted for pathologic examination.

The eye was normal externally. When cut it was evident that there were four small choroidal masses about the disc and one large mass extending out from the

macula to the temporal periphery. Microscopic examination of the globe revealed that there was a malignant melanoma of the ciliary body and the masses noted in the choroid were also found to be malignant melanomas. The predominating cell type was the Callander spindle cell, type B. Elements of spindle cell A and epithelioid cells were also present. Serial sections were made through one of the lesions at the posterior pole and these sections revealed two discrete and independent malignant melanomas. Furthermore, a single benign melanoma of the choroid was discovered in one of the sets of sections. When examined, the patient presented no evidence of other primary malignant melanomas in his body nor was there evidence of metastasis. At the time of this writing the patient is in excellent health.

Pathologic Diagnosis: Multiple malignant melanomas of the choroid, malignant melanoma of the ciliary body, benign melanoma of the choroid, detachment of the retina, drusen of the optic nervehead.

9:10 a.m.

PRESENTATION OF NEW METHODS AND NEW INSTRUMENTS

9:20 a.m.

THE OCULAR EFFECTS OF METHYL ALCOHOL POISONING: REPORT OF A CATASTROPHE INVOLVING THREE HUNDRED TWENTY PERSONS

Curtis D. Benton, Jr., M.D.

BY INVITATION

F. Phinizy Calhoun, Jr., M.D.
Atlanta, Ga.

Discusser:

Albert Potts, M.D.
Cleveland, Ohio

BY INVITATION

Three hundred twenty persons who had consumed varying amounts of bootleg whiskey containing 35 per cent methyl alcohol were examined.

The most useful clinical guide in treatment was the blood carbon dioxide combining power. Not one of the 188 patients whose carbon dioxide was above 25 milliequivalents per liter had visual symptoms or fundus changes. Re-examination two to six months later on 94 of these patients showed all to have remained free from visual damage.

There were 27 deaths. All fatal cases in which a history was obtainable had severe visual loss or blindness before death.

Ninety-five patients with blood carbon dioxide of 25 milliequivalents or less survived. Sixty-five per cent of these patients had early visual symptoms ranging from blurring to blindness. These symptoms were accompanied by visible ophthalmoscopic changes in 87 per cent of these patients. Fundus changes were, first, a hyperemia of the disc which appeared two days after ingestion of methyl alcohol and persisted two to seven days; a peripapillary retinal edema which appeared in three days and remained 10 to 60 days; optic atrophy which became apparent in severely damaged eyes in 30 to 60 days. The initially decreased visual acuity improved in nearly every patient, but again decreased after several weeks in 9 patients whose vision became worse than 20/200 in one or both eyes. All of these 9 patients had initial blood carbon dioxide below 12 milliequivalents.

All patients with dilated, unresponsive pupils either died or had severe visual loss.

The characteristic visual field defect was a cecocentral scotoma.

Six eyes obtained six to eighteen hours after death showed only postmortem degeneration of the retina.

Prolonged and intensive administration of soda bicarbonate is the most important element of treatment. ACTH and spinal fluid exchange therapy were useless.

9:55 a.m.

MECHANICAL FORCES IN INTRACAPSULAR CATARACT EXTRACTION

(A Motion Picture)

Alfred E. Maumenee, M.D.
San Francisco, Calif.

This film was made for teaching purposes. It illustrates the mechanics of the tumbling procedure in intracapsular cataract extraction. The proper application of the intracapsular forceps and the erisophake, and the direction of pull and counterpressure are demonstrated on the eyes of patients and cadavers.

The advantages of grasping the lens near the equator at six o'clock and placing the zonular fibers on stretch before applying counterpressure through the cornea below are stressed.

10:15 a.m.

ACCIDENTAL SURGICAL INJURY TO THE LENS

Leonard Christensen, M.D.
and
Edward McLean, M.D.
BY INVITATION
Portland, Ore.

Discussers:
Edmund B. Spaeth, M.D.
Philadelphia, Pa.
Joseph A. C. Wadsworth, M.D.
New York, N. Y.

The possibility of instrumental injury to the lens was recognized soon after von Graefe introduced the iridectomy in 1857. Since then it has been considered an inherent risk in almost all types of intraocular surgery; however, there has been no systematic attempt to determine the incidence of this complication or evaluate its consequences in contemporary ophthalmic surgery.

In searching for the causes of operative failure in 150 enucleated eyes submitted to the John E. Weeks Memorial Laboratory, histologic evidence of traumatic injury to the lens capsule was found in 20 specimens. Also, gonioscopic and biomicroscopic evidence of accidental injury was found in 7 patients who had had unsuccessful surgery for glaucoma.

These patients have in common certain findings which indicate that peripheral injury to the lens is an important entity which can and should be recognized clinically. The findings in these cases are summarized and discussed from the viewpoint of prevention of this apparently common cause of operative failure in glaucoma.

11:05 a.m.

THERAPEUTIC LAMELLAR CORNEAL GRAFT FOR THE TREATMENT OF HERPETIC OR BULLOUS KERATITIS
(A Motion Picture)

R. Townley Paton, M.D.
New York, N. Y.

This film, which was produced and sponsored by The Eye-Bank for Sight Restoration, Inc., illustrates (1) lamellar corneal graft for treatment of herpetic ulcer; (2) penetrating graft for the removal of a staphyloma; (3) total lamellar transplant in place of keratectomy, and (4) lamellar transplant for the removal of multiple foreign bodies.

11:25 a.m.

OCULAR TOXOPLASMOSIS

Michael J. Hogan, M.D.
San Francisco, Calif.

Discussers:
Helenor Campbell Wilder
Washington, D. C.
Harry A. Feldman, M.D.
Syracuse, N. Y.
BY INVITATION

Congenital toxoplasmosis is accompanied by a chorioretinitis in a high proportion of cases. Many observers have searched for evidence of chronic or latent toxoplasmosis in young and older adults, but as yet definite proof is lacking. A considerable amount of information has accumulated in regard to acute and adult toxoplasmosis, mainly as a result of infections occurring in laboratory workers in this country and abroad. It is not known whether the acute systemic disease is followed by a latent infection in various organs which may become active at a subsequent period. The ease with which latent infections can be produced in laboratory animals suggests that the same thing can occur in the human.

The recent discovery by Wilder that some forms of necrotizing chorioretinitis are due to organisms resembling toxoplasma has added a link in the chain of evidence that acute focal lesions may occur in the body in the absence of active systemic disease. It has not been ascertained whether these acute focal inflammations result from recent infections or are due to reactivation of a latent infection with dissemination of the parasites.

The author has been seeking evidence for the past four years that toxoplasma is or is not a cause of adult human uveitis. As a preliminary to these studies, normal individuals and persons with eye diseases not likely to be caused by toxoplasma were surveyed to determine the incidence of positive reactors in the normal population. Toxoplasmin skin tests and the methylene blue dye test (Sabin-Feldman) were done.

Satisfactory laboratory data were accumulated on 350 cases of uveitis. Complete clinical and laboratory investigations were done on all patients, in addition to the toxoplasmosis studies. The results of these studies will be presented.

In a number of cases the nature of the lesions suggested a possible toxoplasma

infection and serial serologic studies were made. As a result of these investigations a number of uveitis patients have been observed in which a diagnosis of probable ocular toxoplasmosis can be made.

SECTION ON OTOLARYNGOLOGY

Thursday, October 16, 1952
2:00 p.m.

GRAND BALL ROOM

Presiding Officer:
PERCY E. IRELAND, M.D.
Second Vice-President

Secretary:
HOWARD P. HOUSE, M.D.

SOME EXPERIMENTAL OBSERVATIONS BEARING ON MENIERE'S DISEASE
(Wherry Memorial Lecture)
John R. Lindsay, M.D.
Chicago, Ill.

2:30 p.m.
PROPER USE OF ANTIMICROBIAL DRUGS IN ACUTE OTITIS MEDIA
Miriam H. Rutherford, M.D.
Oakland, Calif.

Discussers:
John A. Bigler, M.D.
Chicago, Ill.
BY INVITATION
David D. DeWeese, M.D.
Portland, Ore.

It is a common, tenaciously held opinion that antimicrobials have replaced all other methods of treatment in acute otitis media. This view is erroneous because such treatment often leads to frequent recurrences, spontaneous rupture with scarring of the tympanic membrane and low-grade inflammatory and adhesive changes in the middle ear and mastoid which lead to impairment of hearing.

The object of treatment should be not only to eradicate infection but to restore and preserve hearing. This is best accomplished by adequate drainage of collections of exudate or pus by means of myringotomy, local cleansing, and attention to the underlying causes of infection. Judicious use of antimicrobial drugs as important adjuncts in acute suppurative otitis media is very valuable, but reliance upon such drugs to the exclusion of other treatment is falsely to assume that the tympanic membrane and middle ear have returned to a normal condition merely because of the cessation of pain and fever. Relief of acute symptoms does not constitute a cure. Without drainage, exudate may remain in the middle ear and mastoid and may lead to recurrences or to chronic adhesive changes and consequent hearing impairment. This is especially true in acute catarrhal or secretory otitis media where drainage and maintenance of the normal physiology of the eustachian tube is important to prevent frequent recurrences and chronic adhesive processes.

The antimicrobials are most valuable when used as adjuncts in those cases of acute toxic otitis media in which conservative therapy is not adequate, in impending or early mastoiditis and in those cases in which resolution is delayed. To be effective, the drugs must be bactericidal for the organisms involved and be used in large enough dosage for a period of time sufficient to completely eradicate the infection.

A group of 150 cases of acute otitis media is discussed as illustrative of the foregoing concepts.

3:10 p.m.
ANATOMY AND FUNCTION OF THE TECTORIAL MEMBRANE
Anderson C. Hilding, M.D.
Duluth, Minn.

Discussers:
Edmund P. Fowler, Jr., M.D.
New York, N. Y.
Henry B. Perlman, M.D.
Chicago, Ill.

The true anatomy of the tectorial membrane has never been established. The anatomic relations are of utmost importance in our understanding of the physiology of hearing. Some new points have been worked out and a new theory postulated concerning the manner in which sound vibrations stimulate the hair cells.

3:50 p.m.
TUMORS OF THE NECK
Louis H. Clerf, M.D.
Philadelphia, Pa.

Discussers:
G. O'Neil Proud, M.D.
Kansas City, Kan.
Robert E. Priest, M.D.
Minneapolis, Minn.

Tumors or swellings of the neck often present difficult diagnostic problems. They may represent the first demonstrable evidence of serious disease of the upper air or food passages or be local manifestations of systemic disorders. A multiplicty of causes and a variety of therapeutic procedures that may be indicated suggest that a correct diagnosis be made promptly.

The various conditions that may appear, as swellings of the neck, will be discussed.

4:30 p.m.

ALLERGIC MANAGEMENT OF SOME COMMON EAR CONDITIONS
(Sponsored by the American Society of Ophthalmologic and Otolaryngologic Allergy)
Eugene L. Derlacki, M.D.
and
George E. Shambaugh, Jr., M.D.
Chicago, Ill.

Discussers:
Raymond E. Jordan, M.D.
Pittsburgh, Pa.
Russell I. Williams, M.D.
Cheyenne, Wyo.

That allergic diseases do occur in the ear is not a new concept. However, the lack of one accepted conclusive test to prove an allergy has delayed general acceptance and recognition by the otologist of the frequency of allergic states in the ear.

Our experience has been that therapy-resistant chronic cases of external otitis, of suppurative otitis media of the benign type and of secretory otitis media are frequently allergic in orgin as proved by a response to specific allergic management.

In contrast, a hypersensitivity to a specific inhalant or food is infrequently proved in cases of labyrinthine hydrops, but the excellent results from specific treatment of allergic hydrops justify consideration of a possible allergic etiology in all cases.

SECTION ON OPHTHALMOLOGY

Friday, October 17, 1952
9:00 a.m.

GRAND BALL ROOM

Presiding Officer:
JOHN S. MCGAVIC, M.D.
Third Vice-President

Secretary:
KENNETH L. ROPER, M.D.

HEERFORDT'S SYNDROME
(A Clinicopathologic Case Report)
Georgiana D. Theobald, M.D.
Oak Park, Ill.
and
Howard L. Wilder, M.D.
Chicago, Ill.

BY INVITATION

Female, age 54, with history of parotitis, facial palsy and bilateral uveitis. Both eyes had several operative procedures for secondary glaucoma and the right one ultimately ruptured anteriorly. The pathologic diagnosis: granulomatous iridocyclitis, probably Boeck's sarcoid (Heerfordt's syndrome).

9:10 a.m.

PRESENTATION OF NEW METHODS AND NEW INSTRUMENTS

9:20 a.m.

VISUAL LOSS IN SKULL DEFORMITIES
W. Banks Anderson, M.D.
and
Barnes Woodhall, M.D.
Durham, N. C.

Discusser:
Forrest J. Pinkerton, M.D.
Honolulu, Hawaii

Skull deformities, in themselves of great interest to many branches of science, attract the attention of ophthalmologists because of the occasional occurrence of an accompanying optic atrophy. Because of the rarity of misshapen skulls of high degree in the general population, the etiologic relationship of optic atrophy with the condition may be overlooked. Knowledge of the development of the deformed skull and the associated optic nerve atrophy has accumulated through the overlapping of consecutive waves of

interest on the part of anthropologists, pathologists, geneticists, ophthalmologists, radiologists and more recently the neurosurgeons. Ophthalmic interest, which resulted in many contributions in the late nineteenth century, waned as early surgical intervention not only failed to prevent blindness but was accompanied by a high mortality rate. Poor operative results in the past may have been due, in part, to an erroneous conception of the cause of the atrophy and, in part, to the limitations of the surgical technic of the period.

This paper attempts to trace the development of our knowledge of these misshapen skulls, to point out the difficulties in proper diagnosis in a disease group in which classification is far from satisfactory, to consider those types of deformed skulls in which optic atrophy may be anticipated, and to consider the mechanism of the optic atrophy. The major points in modern surgical technic directed towards the protection of vision in these cases will be described. The indications for operation and the postoperative results in a limited number of cases will be reviewed.

9:55 a.m.

CATARACT EXTRACTION WITH THREE TRACK SUTURES

(A Motion Picture)

Brendan D. Leahey, M.D.
Lowell, Mass.

Lid pressure on the eyeball is completely avoided by means of an Arruga speculum. A superior rectus suture is inserted. A small conjunctival flap is dissected up from the limbus. Three 5-0 silk track sutures of Verhoeff type are inserted in the superficial episclera and cornea at the limbus at eleven, twelve and one o'clock. These tracks are stained with methylene blue and the sutures left in place. The eye is immobilized with a Holmes double fixation forceps grasping the conjunctiva at 3:30 and 8:30, close to the limbus. Corneal section is made through the track sutures. Silk 6-0 atraumatic sutures are rethreaded through these tracks and the remnants of original track sutures are discarded. The sutures come out through the conjunctiva in mattress fashion. The corneal section is enlarged slightly with McGuire scissors. The wound is opened by means of traction on the middle suture. A four-cut iridectomy gives a narrow keyhole coloboma. A diagram of the iridectomy technic is included. The lens is grasped just in front of the equator at twelve o'clock, dislocated above, and slid out by Verhoeff's technic mainly from pressure inferiorly. Sutures are tied. Iris is smoothed out and an air bubble is inserted in anterior chamber.

10:15 a.m.

ALBINISM

Harold F. Falls, M.D.
Ann Arbor, Mich.

Discusser:
John Clement McCulloch, M.D.
Toronto, Ont., Canada

Albinism, an inborn error of melanin pigment formation, has intrigued the curiosity and attention of the physician as well as the laity since the beginning of recorded history. The author will undertake to review and summarize the literature on the subject as well as to present original research findings. The ocular consequences of albinism include photophobia, nystagmus, error of refraction, reduced visual acuity, central scotoma and varying degrees of absence of retinal pigmentation. The complex will be analyzed from the viewpoint of diagnosis, prognosis, and genetics. Many different genotypes effect albinism of the eye and specific pedigrees and photographs will be utilized to illustrate their variations in expressivity.

Partial albinism associated with phenylpyruvica oligophrenia, Waardenburg's syndrome and other syndromes will be discussed.

10:50 a.m.

AN ORBITAL IMPLANT OF PLASTIC SPONGE

(A Motion Picture)

Maurice D. Pearlman, M.D.
Chicago, Ill.

A demonstration of technics and postoperative results wherein "Ivalon," an inert polyvinyl sponge, is implanted retroconjunctivally following enucleation.

11:10 a.m.

FURTHER OBSERVATIONS ON THE SYNDROME OF GLAUCOMATOCYCLITIC CRISES

Adolph Posner, M.D.
Abraham Schlossman, M.D.
New York, N. Y.

Discussers:

Peter C. Kronfeld, M.D.
Chicago, Ill.
Harold G. Scheie, M.D.
Philadelphia, Pa.

Since our original description of this syndrome in 1948, we have been impressed by the frequent occurrence of this type of glaucoma. Its recognition is important, not only for the management of the specific case, but also for the differential diagnosis of the glaucomas in general and, especially, acute narrow angle glaucoma.

Recent observations by us and reports from other ophthalmologists have confirmed the original impression that the syndrome follows a uniform pattern, both as to symptomatology and the clinical course.

The syndrome of glaucomatocyclitic crises is a recurrent unilateral acute glaucoma, not of the narrow angle type, and usually associated with signs denoting the presence of a mild cyclitis, such as cells in the aqueous and a few keratic precipitates.

The elevation of tension is out of proportion to the inflammatory reaction. The individual attacks are self-limited, but the total duration may extend over many years. The treatment of choice consists of mild mioties and cortisone. Surgical procedures have not proved of value in preventing recurrences.

11:55 a.m.

AN IMPROVED MOTILITY IMPLANT
(A Motion Picture)

Max Fine, M.D.
San Francisco, Calif.

Surveys of the late results from various types of orbital implants after enucleation, conducted by this society (1951) and others, indicate that an ideal implant combining maximal motility with a minimum of complications is not yet available. Semiburied integrated implants give the best cosmetic results but are too often displaced, extruded or productive of excessive discharge. The simple spherical implant into Tenon's capsule has few complications but transmits insufficient movement to the prosthesis.

The motion picture depicts the technic of inserting a tunnelled, buried, magnetic implant. It consists of a machined lucite sphere, 16 mm. in diameter, with flattened, obliquely directed channels for the rectus tendons. Sharp edges which erode tissue have been eliminated. The anterior face is flattened and has an Alnico permanent magnet embedded 1.0 mm. below the surface. The implant is covered by the tendons, Tenon's capsule, and conjunctiva. The tissues are in contact with the plastic only. A second magnet is embedded beneath the posterior surface of the plastic prosthesis.

No originality is claimed for this device. The advantageous features of many pre-existing implants have been modified and combined into what is believed to be a practical working compromise between the integrated implant and the simple buried sphere. Fifty such implants have been placed in the past two years by several surgeons on the Pacific Coast. This period of observation is too short, but it is promising that there have been no extrusions and no instances of excessive discharge. Motility averages 60 per cent of normal.

12:15 p.m.

INTRAOCULAR ACRYLIC LENSES

Mr. Harold Ridley
London, England

BY INVITATION

Discusser:

Derrick T. Vail, M.D.
Chicago, Ill.

An account is given of the development of intraocular acrylic lenses after cataract extraction.

These lenses restore the eye to normal except for loss of accommodation and complete the cure for cataract which is only half treated by plain extraction. A full visual field without distortion is obtained, and should the other eye be normal, or nearly so, binocular vision is restored.

Some 60 cases are reviewed, which show that the lens is well tolerated for at least two and a half years and that excellent acuity is obtainable. The technic is described and difficulties discussed.

SPECIAL SCIENTIFIC PROGRAMS

OTOSCLEROSIS STUDY GROUP

Division of
Committee on Conservation of Hearing

Private Dining Room 14
Saturday, 8:00 p.m.

PHILIP E. MELTZER, M.D.
President

THEODORE E. WALSH, M.D.
Secretary

Does Otosclerosis Cause Cochlear Nerve Degeneration?
Stacy R. Guild, Ph.D., Baltimore, Md.

Observations on Experimentally Produced Perceptive Deafness
Harold F. Schuknecht, M.D., Chicago, Ill.

Evaluation of Technical Factors Introduced for the Improvement of Fenestration Surgery
Julius Lempert, M.D., New York, N. Y.
and
Philip E. Meltzer, M.D., Boston, Mass.

TEACHERS' SECTION — OTOLARYNGOLOGY

Private Dining Room 14
Sunday, 10:00 a.m.

LAWRENCE R. BOIES, M.D., *Chairman*

Discussion of Dr. Philip E. Meltzer's paper, "What Should the Clinical Otologist Do With 'Audiology'?"
Frederick T. Hill, M.D., Waterville, Me.
George E. Shambaugh, Jr., M.D., Chicago, Ill.

General Discussion

Full Time vs. Part Time Teaching in Otolaryngology

The Viewpoint of the Full Time Teacher
John A. Kirchner, M.D., New Haven, Conn.

The Viewpoint of the "Geographical" Full Time Teacher
Frank S. Forman, M.D., Little Rock, Ark.

The Viewpoint of the Part Time Teacher
Albert C. Furstenberg, M.D., Ann Arbor, Mich.

Is a Rotating Internship Important Toward Interesting Physicians in Our Specialty?
Stanton A. Friedberg, M.D., Chicago, Ill.

TEACHERS' SECTION — OPHTHALMOLOGY

Crystal Room
Sunday, 10:00 a.m.

A. D. RUEDEMANN, M.D., *Chairman*

The Home Study Course: Its Value and Place in Graduate Education
Hendrie W. Grant, M.D., Minneapolis, Minn.

DISCUSSER:
W. L. Benedict, M.D., Rochester, Minn.

Formal Basic Courses in Ophthalmology
Edward B. Spaeth, M.D., Philadelphia, Pa.

DISCUSSER:
Lawrence T. Post, M.D., St. Louis, Mo.

Clinical Instruction in Ophthalmology with Isolated Courses on Special Subjects
Alan C. Woods, M.D., Baltimore, Md.

DISCUSSER:
F. Bruce Fralick, M.D., Ann Arbor, Mich.

Intensive Short Courses such as the Lancaster Course
James J. Regan, M.D., Boston, Mass.

DISCUSSER:
Derrick Vail, M.D., Chicago, Ill.

COMMITTEE ON CONSERVATION OF HEARING
and
OTOSCLEROSIS STUDY GROUP DIVISION

Grand Ball Room
Sunday, 8:00 p.m.

DEAN M. LIERLE, M.D., *Chairman*

PHILIP E. MELTZER, M.D., *Co-Chairman*

How to Get a Conservation of Hearing Program Started
Mrs. Lydia Newton, Program Consultant
National Society for Crippled Children and Adults, Phoenix, Ariz.

How to Organize Your State Medical Association for a Conservation of Hearing Program
Joseph M. Kinkade, M.D., Chairman
State Hearing Conservation Committee of the State Medical Society, Tucson, Ariz.

Description of the Educational Follow-Up in a Conservation of Hearing Program
Archie E. Cruthirds, M.D., Chairman
Hearing Conservation Committee, Maricopa County Medical Society, Phoenix, Ariz.

Conservation of Hearing
Edmund Prince Fowler, Sr., M.D., New York, N. Y.

INDUSTRIAL OPHTHALMOLOGY

Private Dining Room 14
Sunday, 8:00 p.m.

A. D. RUEDEMANN, M.D., *Chairman*
HEDWIG S. KUHN, M.D., *Secretary*

Visual Problems in Industry
Henry Imus, Ph.D.
Office of Naval Research, Washington, D. C.

The Eye Clinic at Oak Ridge National Laboratory
Jean S. Felton, M.D., Medical Director
Oak Ridge National Laboratory
Oak Ridge, Tenn.

The Ophthalmologist and the Insurance Company
Carl T. Olson, M.D., Medical Director
Liberty Mutual Insurance Company, Midwest Section, Chicago, Illinois

Presentation of the Annual Joint Committee Award to
Albert C. Snell, Sr., M.D.
Rochester, N. Y.

COMMITTEE ON PLASTIC SURGERY

Grand Ball Room
Tuesday, 8:00 p.m.

JOHN MARQUIS CONVERSE, M.D., *Chairman*

(Program to be announced.)

AMERICAN ORTHOPTIC COUNCIL
and
AMERICAN ASSOCIATION OF ORTHOPTIC TECHNICIANS

Private Dining Room 14
Tuesday, 8:00 p.m.

FRANK D. COSTENBADER, M.D.
Secretary

SYMPOSIUM:
CONVERGENCE INSUFFICIENCY

Hermann M. Burian, M.D.
Moderator
Iowa City, Iowa

Physiopathology
Walter H. Fink, M.D., Minneapolis, Minn.

Diagnosis and Incidence
Nancy M. Capobianco, New York, N. Y.

Medical Management
G. Victor Simpson, M.D., Washington, D. C.

Orthoptic Treatment
Electra Healy, Chicago, Ill.

OPHTHALMIC ANATOMY AND EMBRYOLOGY

Motion Pictures

Grand Ball Room
Thursday, 8:00 p.m.

JOHN S. MCGAVIC, M.D., *Chairman*
KENNETH L. ROPER, M.D., *Secretary*

Anatomy of the Orbit
W. H. Hollinshead, Ph.D., Rochester, Minn.

Ocular Biomicroscopy
Milton L. Berliner, M.D., New York, N. Y.

The Embryology of the Eye
Harold F. Falls, M.D., Ann Arbor, Mich.

AMERICAN SOCIETY OF OPHTHALMOLOGIC and OTOLARYNGOLOGIC ALLERGY

Private Dining Room 14
Friday, 9:00 a.m.

HUGH A. KUHN, M.D.
President

JOSEPH W. HAMPSEY, M.D.
Secretary

Introductory Remarks
Hugh A. Kuhn, M.D., Hammond, Ind.

9:10 a.m.—Pathology of Allergic Rhinitis and Sinusitis
Herman Semenov, M.D., Beverly Hills, Calif.

10:00 a.m.—Autonomic Nervous System
Benjamin Spector, M.D., Boston, Mass.

11:00 a.m.—The Common Ground of Allergy, Autonomic Dysfunction and Endocrine Imbalance
Jerome Hilger, M.D., St. Paul, Minn.

12:30 p.m.—Luncheon Meeting

Round Table
Vascular Headaches and Related Phenomena
French K. Hansel, M.D., St. Louis, Mo.

Grand Ball Room
Friday, 2:00 p.m.

Pathological Findings in Allergic Eye Diseases
Brittain F. Payne, M.D., New York, N. Y.

Allergy of the Cornea and Bulbar Conjunctiva
A. D. Ruedemann, M.D., Detroit, Mich.

Diagnosis and Treatment in Ocular Allergy
S. Albert Hanser, M.D., St. Louis, Mo.

Ocular Allergy Illustrated by Kodachrome Slides
Harry Stauffer, M.D., Jefferson City, Mo.

3:30 p.m.—Oral Lesions in Allergy
Charles K. Shofstall, M.D., Kansas City, Mo.

SCIENTIFIC EXHIBITS

RED LACQUER ROOM
Kenneth L. Roper, *Chairman*
Chicago, Ill.

EXHIBIT S-1
AMERICAN BOARD OF OPTICIANRY

Exhibitor:
Philip E. Dempsey
BY INVITATION
Chicago, Ill.

This exhibit consists of charts and diagrams concerning the purposes, functions and work of the American Board of Opticianry. The charts and diagrams contain information concerning (1) educational requirements, curricula and schools of opticianry accredited by the Board and (2) certification by the Board through examinations for Associate in and Master of Ophthalmic Optics.

EXHIBIT S-2
MEDICAL PHOTOGRAPHY

Exhibitor:
Maria Ikenberg
BY INVITATION
Biological Photographic Association, Inc.
Chicago, Ill.

The exhibit will consist of a unit covering the back wall of the booth under the heading: "Medical Illustration—A Modern Aid to Medicine." The unit is subdivided in panels, each depicting one specific phase of the subject. The side walls will show a few selected photographs representative of lighting technics applied in medical photography. The booth will be attended by medical photographers who will be prepared to answer questions concerning equipment and methods employed.

EXHIBIT S-3
BLINDNESS AMONG PRESCHOOL CHILDREN

Exhibitor:
Franklin M. Foote, M.D.
BY INVITATION
National Society for the Prevention of Blindness
New York, N. Y.

Co-Exhibitor:
Virginia Smith Boyce
BY INVITATION

This exhibit is based on a study of the prevalence and causes of blindness among infants and preschool children in twenty states. It consists of statistical charts, posters, drawings and maps.

EXHIBIT S-4
THE OCULAR PATHOLOGY OF FOREIGN BODIES

Exhibitors:
Bernard A. Roberts, M.D.
and
Abraham Schlossman, M.D.
Department of Pathology, New York Eye and Ear Infirmary
New York, N. Y.

Co-Exhibitor:
Edgar B. Burchell, D. Sc.

Color photomicrographs of pathologic sections showing iron, wood, cilia, lead and copper in situ will be presented. Tissue reactions in the early and late stages will be demonstrated. Several kodachrome transparencies of gross specimens showing the presence of copper in the tissues, stone and iron in the vitreous and lead in the optic nerve will be presented. In addition there will be a small panel showing the early and late effects of radiation on the ocular tissues.

EXHIBIT S-5
ANATOMY OF THE TRACHEOBRONCHIAL TREE

Exhibitor:
Bernard S. Kleiman, M.D.
Department of Anatomy, University of Maryland Medical School
Baltimore, Md.

Two models are shown, one a direct metallic cast of the tracheobronchial tree and the second, a four-foot copper tubing copy of the direct cast. Six descriptive colored charts are also used. The colored charts and the models have been colored to demonstrate the various bronchopulmonary segments.

EXHIBIT S-6
SPEECH AND HEARING REHABILITATION

Exhibitor:
Lawrence J. Linck
BY INVITATION
National Society for Crippled Children and Adults, Inc.
Chicago, Ill.

Co-Exhibitors:
Dean M. Lierle, M.D.
and
Jayne Shover, Lydia Newton, Herbert Koepp Baker, Ph.D., and Harold Westlake, Ph.D.
BY INVITATION

The exhibit will describe the nationwide work of the Easter Seal society on behalf of the speech and hearing handicapped.

EXHIBIT S-7
NOSEBLEED AND ITS MANAGEMENT

Exhibitor:
O. E. Hallberg, M.D.
Mayo Clinic
Rochester, Minn.

Severe nosebleed is mainly a geriatric problem. Location of site of bleeding is of utmost importance in its treatment. This exhibit (1) demonstrates the blood supply of the nose with special emphasis upon the external and internal carotid systems, (2) evaluates the various causes of nosebleed and (3) describes methods of treatment such as (a) electrocoagulation, (b) spot packing, (c) postnasal packing, (d) ligation of arteries, (e) submucous resection and (f) supportive measures such as blood transfusions and treatment with vitamin C, vitamin K and rutin.

EXHIBIT S-8
GLAUCOMA: INTRAMEDICAL COOPERATION FOR CASE FINDING AND REFERRAL

Exhibitor:
Conrad Berens, M.D.
The Ophthalmological Foundation, Inc.
New York, N. Y.

Co-Exhibitor:
Charles P. Tolman
BY INVITATION

The exhibit comprises lecture and instructional material to enlist cooperation of other physicians with ophthalmologists in their respective areas for instruction by the ophthalmologists as preceptor in early recognition of ocular disease, particularly glaucoma. Clinical and other instruction will be given in the use of a special "screening" tonometer which enables the physician to make an objective test for ocular hypertension. A comparison manometer will also be shown.

EXHIBIT S-9
CARCINOMA OF THE PARANASAL SINUSES

Exhibitor:
Harold F. Schuknecht, M.D.
University of Chicago Clinics
Chicago, Ill.

Co-Exhibitor:
Robert M. Appleman, D.D.S.
BY INVITATION

The surgical and prosthetic management of carcinoma of the paranasal sinuses will be demonstrated by drawings, preoperative and postoperative photographs, colored pictures of specimens and models. The exhibit emphasizes the importance of a thorough surgical procedure followed by appropriate radiation therapy. The postoperative management of the patient is greatly facilitated by the use of proper oral prostheses.

EXHIBIT S-10
THE FENESTRATION OPERATION: THE CALIBRATED WEBER TEST, POSTOPERATIVELY

Exhibitor:
J. Brown Farrior, M.D.
Southern Foundation for Better Hearing
Tampa, Fla.

Co-Exhibitor:
Richard A. Bagby, M.D.
BY INVITATION

The calibrated Weber test determines the degree of postoperative serous labyrinthitis. Performed with the bone conduction audiometer, variations in lateralization are affected by recruitment and the severity of the postoperative reaction in the inner ear. The routine daily calibration of the lateralization of bone conduction enables the surgeon to evaluate the resultant inner ear response to minute variations in surgical technic. More accurate than the tuning fork, the calibrated

Weber test is of prognostic value in determining the eventual outcome of a fenestration operation.

EXHIBIT S-11

HEADACHE: ITS DIFFERENTIAL DIAGNOSIS AND TREATMENT

Exhibitor:
Robert E. Ryan, M.D.
St. Louis, Mo.

The exhibit will consider the following aspects of headache: (1) the physiologic basis of head pain, (2) the history of the patient who has headaches, (3) the objectives of symptomatic and prophylactic forms of treatment, (4) the examination of the patient with headache, (5) various types of headache with their symptoms, signs and treatment, such as migraine, histaminic cephalgia, sinusitis, myalgia, tension, hypertension, psychogenic, otic and ocular, and (6) the results obtained with experimental preparations in treating headache.

EXHIBIT S-12

ROENTGENOGRAPHIC CONTRIBUTIONS TO OCULAR DIAGNOSIS

Exhibitors:
Maurice D. Pearlman, M.D.
and
Eugene R. Elstrom, R.T.
BY INVITATION
University of Illinois, Illinois Eye & Ear Infirmary
Chicago, Ill.

This is a display and description of films which aid in establishing the diagnosis in various congenital, inflammatory, neoplastic, traumatic and degenerative conditions of the eye and adnexa. The exhibit also will describe various radiographic technics and display several armamentaria utilized in such work.

EXHIBIT S-13

ORTHOPTICS

Exhibitor:
Ann Stromberg, Chairman
BY INVITATION
American Association of Orthoptic Technicians

Co-Exhibitor:
Department of Ophthalmology,
State University of Iowa
Iowa City, Iowa

What can the ophthalmologist without a technician do for the orthoptic patient? He can (1) test the status of binocular vision (schematic drawings and pictures), (2) select cases for orthoptic treatment (including occlusion): preoperative, postoperative, or without surgical intervention, (3) determine the possibilities and limitations of various orthoptic instruments, (4) stress the importance of parents' insight into problems and objectives of simple home exercises and (5) suggest methods of recording binocular findings at first and subsequent visits.

EXHIBIT S-14

HEARING IS PRICELESS

Exhibitor:
Ada M. Hill
BY INVITATION
American Hearing Society
Washington, D. C.

The exhibit will consist of posters and photographs and an automatic slide machine with 30 slides depicting services for the hearing handicapped. Information will be available on the prevention of deafness, conservation of hearing, and rehabilitation of the hard of hearing. There will be daily demonstrations of lipreading and the use of hearing aids.

EXHIBIT S-15

THE PHYLOGENY AND ONTOGENY OF OTORHINOLARYNGOLOGY

Exhibitor:
Lyle M. Sellers, M.D.

Co-Exhibitor:
Ludwig A. Furchgott, M.D.

The story of otorhinolaryngology as a step in the cultural and scientific development of man is illustrated by the exhibition and description of selected illuminated manuscripts and books responsible for the dissemination in the western world of cultural and general scientific and medical knowledge. Out of this background is shown the development of otorhinolaryngology as a science, further illustrated by

the exhibition and description of a first or a very early edition of most of the published mileposts in the history of this specialty.

EXHIBIT S-16

ATLAS OF OTOLARYNGIC PATHOLOGY

Exhibitor:
The American Registry of Pathology of the Armed Forces Institute of Pathology
Washington, D. C.

Transparencies illustrating the organization, contents and sample gross and microscopic photographs of the new Atlas of Otolaryngic Pathology are shown. This atlas is sponsored by the Academy in conjunction with the American Registry of Pathology of the Armed Forces Institute of Pathology.

EXHIBIT S-17

ATLAS OF OPHTHALMIC PATHOLOGY

Exhibitor:
The American Registry of Pathology of the Armed Forces Institute of Pathology
Washington, D. C.

Transparencies illustrating the organization, contents and sample gross and microscopic photographs of the new Textbook-Atlas of Ophthalmic Pathology are shown. This atlas is sponsored by the Academy in conjunction with the American Registry of Pathology of the Armed Forces Institute of Pathology.

EXHIBIT S-18

FAILURES IN FENESTRATION SURGERY

Exhibitor:
George E. Shambaugh, Jr., M.D.
Northwestern University Medical School
Evanston, Ill.

Co-Exhibitors
Eugene L. Derlacki, M.D.
and
Donald W. Shuster, M.D.
Jesse E. Waller, M.D.

BY INVITATION

Failures in fenestration surgery are defined and analyzed in 3,000 consecutive operations. The important causes for failure are osteogenic closure, improper selection of cases and postoperative serous labyrinthitis. The factors that influence these failures are described and methods of prevention are detailed.

EXHIBIT S-19

SURGICAL MANAGEMENT OF CHRONIC MASTOIDITIS

Exhibitor:
Kinsey M. Simonton, M.D.
Mayo Clinic
Rochester, Minn.

The exhibit consists of models which illustrate the surgical management of chronic mastoiditis and its common complications. Exposure of the surgical field by both endauricular and postauricular routes will be demonstrated. The indications and technic of radical mastoidectomy and modified radical mastoidectomy will be given. Models will demonstrate the gross pathology and the surgical procedures indicated in cases of chronic infection of the mastoid cells, cholesteatosis of the epitympanic space and mastoid antrum, phlebitis of the sigmoid sinus, thrombosis of the sigmoid sinus, pachymeningitis, subdural abscess or contiguous brain abscess, fistula of the semicircular canal, suppurative labyrinthitis, abscess of the petrous pyramid and inflammation involving the facial nerve.

EXHIBIT S-20

PREVENTIVE OPHTHALMOLOGY

Exhibitor:
Isadore Givner, M.D.
New York Eye & Ear Infirmary, New York University, Bellevue Medical Center
New York, N. Y.

Co-Exhibitor:
Bernard Kronenberg, M.D.

Physicians have an opportunity to prevent certain eye diseases. These will be grouped according to the various medical specialties.

EXHIBIT S-21

A. SURGICAL TREATMENT OF CHRONIC FRONTAL SINUSITIS BY USE OF SEWALL MUCOPERIOSTEAL FLAP

B. INTRANASAL DACRYOCYSTORHINOSTOMY

Exhibitor:
Guy L. Boyden, M.D.
University of Oregon Medical School
Portland, Ore.

Co-Exhibitors:
Lester T. Jones, M.D.
Robert M. Hansen, M.D.

A. A mucoperiosteal flap is dissected from the lateral wall of the nose and hinged at the nasal fissure to create permanent drainage of the frontal sinus.

B. Surgical technic describing intranasal dacryocystorhinostomy.

EXHIBIT S-22
OTOTOXIC EFFECTS OF STREPTOMYCIN AND THE ALLIED ANTIBIOTICS

Exhibitor:
Aram Glorig, M.D.
George Washington University
School of Medicine
Washington, D. C.

Co-Exhibitor:
Joseph E. Hawkins, Ph.D.
Merck Institute for Therapeutic Research
Rahway, N. J.

BY INVITATION

A presentation of data from human and animal studies regarding the lesion and its location, the type and amount of dysfunction caused by streptomycin and the allied antibiotics. The exhibit will show some new types of auditory testing equipment.

EXHIBIT S-23
HEMANGIOMAS: TYPES AND METHODS OF TREATMENT

Exhibitor:
Frederick A. Figi, M.D.
Mayo Clinic
Rochester, Minn.

Co-Exhibitor:
John B. Erich, M.D.

This exhibit presents a systematic review of hemangiomas and a detailed discussion of the methods of treatment which have been found most effective for each type of these vascular lesions. Emphasis is placed on the response to irradiation of each type of hemangioma and the serious consequences which can result from the injudicious use of radium or roentgen therapy.

EXHIBIT S-24
THE NEWLY BLIND PATIENT

Exhibitor:
Helen Keller
BY INVITATION
The American Foundation
for the Blind, Inc.
New York, N. Y.

Co-Exhibitors:
M. Robert Barnett
Kathern F. Gruber

BY INVITATION

The American Foundation for the Blind believes that the ophthalmologist is a powerful influence with a newly blind patient. Literature exhibited will give information concerning tools and aids for the blind, rehabilitation services available, help for parents of blind babies, attitudes toward blindness, etc. Professional staff members of the Foundation will be in attendance at the booth at all times.

EXHIBIT S-25
CORRELATION OF GROSS ANATOMY AND X-RAY FINDINGS IN MISCELLANEOUS EAR, NOSE AND THROAT LESIONS: CLINICOPATHOLOGIC STUDY USED FOR TEACHING

Exhibitor:
George I. Uhde, M.D.
Departments of Otolaryngology and
Pathology of Louisville V. A. Hospital
and University of Louisville
School of Medicine
Louisville, Ky.

Co-Exhibitors:
Harold Gordon, M.D.
Heinz Oppenheim, M.D.

BY INVITATION

The purpose of this collection is to illustrate a method of teaching gross pathology with x-ray findings to medical students and also residents seeking certification in otolaryngology. Microscopic sections of the specimens exhibited are available but not shown because we believe the resident must examine most of the slide to obtain a proper understanding of the lesion.

EXHIBIT S-26
HOARSENESS

Exhibitor:
James Julian O'Neill, M.D.
Creighton University School of Medicine
Omaha, Neb.

A mirror image of a larynx in motion with two revolving drums demonstrates the organic pathology of the larynx causing hoarseness. This pathology is shown in colored drawings of the larynx and each is accompanied by a brief clinical resume of the particular case. There are eighteen illustrated cases thus presented.

EXHIBIT S-27
MODELS OF GONIOSCOPIC VIEWS OF THE ANTERIOR CHAMBER ANGLE

Exhibitor:
Arthur Shainhouse, M.D.
BY INVITATION
Glaucoma Clinic
Brooklyn Eye and Ear Hospital
Brooklyn, N. Y.

The exhibit consists of a series of ceramic models demonstrating the gonioscopic findings of the angles of the anterior chamber of eyes of patients studied in the Glaucoma Clinic of the Brooklyn Eye and Ear Hospital. Patients with normal and pathologic eyes were studied by members of the Clinic and models were made by the exhibitor of some of the more interesting and illustrative angles. Particular attention is paid to such conditions as glaucoma and complications following various intraocular operations.

EXHIBIT S-28
BACTERIOLOGIC AID IN RHINOLOGIC DIAGNOSIS AND TREATMENT

Exhibitor:
Joseph L. Goldman, M.D.
The Mount Sinai Hospital and New York University Post Graduate Medical School
New York, N. Y.

Transparencies of charts, color photomicrographs and case reports show the importance of bacteriologic and cytologic examinations of nasal and sinus secretions in rhinologic diagnosis and treatment. The value of such data as major criteria in the differential diagnosis of infectious and noninfectious (vasomotor) disorders is presented. The need to determine the susceptibility of micro-organisms isolated from infections to antibiotics before instituting such therapy is indicated.

EXHIBIT S-29
HOLE FORMATION IN THE PERIPHERAL RETINA

Exhibitor:
C. C. Teng, M.D.
BY INVITATION
The Eye Bank for Sight Restoration, Inc.
New York, N. Y.
Co-Exhibitor:
Herbert M. Katzin, M.D.

The exhibit depicts the mechanism of hole formation in the peripheral retina. "Normal eyes" were examined by the dissecting microscope and serial sections made of the lesions found. The present study is concerned with hole formation from congenital rosettes and from peripheral cystoid degeneration.

TECHNICAL EXHIBITORS — 1952

Exhibitor	Booth No.
Abbott Laboratories	106 & 107
Alcon Laboratories, Inc.	57
Alkalol Co.	29
American Cystoscope Makers, Inc.	91 & 92
American Optical Co.	7 thru 14
Audiometer Sales Corp.	47
Aurex Corp.	R-3
Barnett Optical Laboratories	37
Bausch & Lomb Optical Co.	87 thru 90 & 95 thru 98
Bay State Optical Co.	105
Austin Belgard, Inc.	18 & ½ of 19
Beitler-McKee Optical Co.	103
Bell & Howell Co.	58
N. P. Benson Optical Co.	84 & 85

Exhibitor	Booth No.
Blakiston Co.	83
Boll & Lewis Optical Co.	41
Burroughs Wellcome & Co., Inc.	R-4
Geo. H. Butterfield & Son	R-9
Camel Cigarettes	27 & 28
Cameron Surgical Specialty Co.	74
Ciba Pharmaceutical Products, Inc.	R-10
ClerSite Co.	"C"
Coca-Cola Co.	26
Coreco Research Corp.	59
Davis & Geck, Inc.	R-6
DeVilbiss Co.	15 & 16
Doho Chemical Corp.	34
Encyclopaedia Britannica, Inc.	R-7
Grieshaber Swiss Eye Instruments	38
Grune & Stratton, Inc.	35
Guild of Prescription Opticians of America, Inc.	43 & 44
Paul B. Hoeber, Inc.	32
Hollister-Stier Laboratories	46
The House of Vision, Inc.	60, 61 & 62
Ion Optical Co.	R-5
Keeler Optical Products, Ltd.	77
Keystone View Co.	23 & 24
Kono Manufacturing Co.	76
Lea & Febiger	51
J. B. Lippincott Co.	R-8
Mager & Gougleman, Inc.	20 & ½ of 19
Maico Co., Inc.	66 & 67
Mann Instrument Co.	R-17
Marcelle Cosmetics, Inc.	54
Medical Case History Bureau	112
Medical Film Guild, Ltd.	"A"
Merck & Co., Inc.	110
E. B. Meyrowitz Surgical Instruments Co., Inc.	79 & 80
Modern Optics, Inc.	108 & 109
C. V. Mosby Co.	86
V. Mueller & Co.	1 thru 6
New Era Optical Co.	R-19 & R-20
New Jersey Optical Co.	48
Novocol Chemical Manufacturing Co., Inc.	68
Obrig Laboratories, Inc.	102
Paravox, Inc.	R-13
Chas. Pfizer & Co., Inc.	R-14
Philip Morris & Co., Ltd., Inc.	75
George P. Pilling & Son Co.	63, 64 & 65
Precision-Cosmet Co., Inc.	"B"
Professional Press, Inc.	50
Radium Chemical Co., Inc.	30
Ritter Co., Inc.	69 thru 73
W. B. Saunders Co.	49
Schering Corp.	33
Scincy Co.	R-1
Shuron Optical Co., Inc.	81 & 82
Smith, Kline & French Laboratories	45
Soft-Lite Lens Co., Inc.	104
Sonotone Corp.	78
C. M. Sorensen Co.	42
Storz Instrument Co.	99, 100 & 101
Sturgis-Grant Productions, Inc.	R-2
Surgical Mechanical Research	93 & 94
Thermosector Co.	31
Titmus Optical Co., Inc.	56
Travenol Laboratories, Inc. (Subsidiary of Baxter Laboratories, Inc.)	R-21 & R-22
Uhlemann Optical Company of Illinois	52 & 53
Univis Lens Co.	39 & 40
Victory Optical Manufacturing Co.	111
Visionez Corp.	R-12
Dr. Max M. von Stirne	R-18
Welch Allyn Co.	17
White-Haines Optical Co.	21 & 22
Williams & Wilkins Co.	36
Winthrop-Stearns, Inc.	25
Carl Zeiss, Inc.	R-16
Zenith Radio Corp.	55
Zylite Products Co., Inc.	R-11
Zylo Ware Corp.	R-15

TECHNICAL EXHIBITS

The Academy is appreciative of the cooperation of all technical exhibitors.

Booths with numbers preceded by "R" are located in the Red Lacquer Room.

Abbott Laboratories, North Chicago, Ill. Booths 106 & 107

Abbott Laboratories will exhibit two important products: PENTOTHAL SODIUM, an ultra-short-acting barbiturate for intravenous anesthesia, and DAYALETS, a multiple vitamin tablet with no fish-oil taste. The DAYALETS exhibit illustrates the absence of fish-oil burp and allergic reactions in the product.

Alcon Laboratories, Inc., Fort Worth, Texas Booth 57

Alcon Laboratories, Incorporated, is exhibiting a new methyl cellulose ophthalmic solution (Isopto Alkaline) as a vehicle and soothing ophthalmic lubricant —along with other ophthalmic and nasal drops—uniformly buffered for optimal effect and patient acceptance.

Alkalol Co., Taunton, Mass. Booth 29

The Alkalol Company is featuring ALKALOL, a solution especially designed for the treatment of the mucous membranes of the eye, nose and throat.

First compounded in 1896, its balanced formula of salts and essential oils makes a solution that is effective and at the same time nontoxic, mild and pleasant. It is alkaline and a mucous solvent.

ALKALOL is an excellent vehicle for many drugs. For further information, please call at Booth 29.

American Cystoscope Makers, Inc., New York, N. Y. Booths 91 & 92

Precision instruments for diagnosis and operative procedures will be on display in our booth. These include nasopharyngoscopes of advanced design, thoracic electroresection equipment, low-reflection treated telescopes for bronchoscopy and esophagoscopy, together with equipment for endoscopic photography. Representatives will be in attendance to demonstrate these and to answer any questions which may arise. A cordial invitation is extended to members and guests of the Academy.

American Optical Co., Southbridge, Mass. Booths 7 thru 14

The American Optical Company, Instrument Division, will have a complete display of refracting and diagnostic instruments including the new Schepens Indirect Binocular Ophthalmoscope and the new Ful-Vue line. Orthoptic instruments and equipment including the Walraven Bar Separator will also be demonstrated. In addition, there will be on display a selection of laboratory instruments of interest to the professions.

Audiometer Sales Corp., Minneapolis, Minn. Booth 47

Aurex Corp., Chicago, Ill. Booth R-3

Barnett Optical Laboratories, Chicago, Ill. Booth 37

Innovations in the field of photography of the anterior segment of the eye will be on exhibit.

A new camera that can take single or stereoscopic pictures will be the headliner of this display, which will also include other optical and photographic specialties, such as a new electronic flash unit for eye work, copying devices, head loupes, magnifiers, pocket and small stand microscopes, a new student microscope, slide libraries of eye diseases and injuries, slide viewers and projectors.

Bausch & Lomb Optical Co., Rochester, N. Y. Booths 87 thru 90 & 95 thru 98

Bay State Optical Co., Attleboro, Mass. Booth 105

Bay State will display a complete line of ophthalmic frames, featuring several different types of the BSO-created HEIRESS. In addition, many new trims for the CHIQUITA frame will be shown. In attendance at the Bay State booth will be George F. Pearce, Sales Manager, Fred Slosson and Al Kohler.

Austin Belgard, Inc., Chicago, Ill. Booth 18 & ½ of 19

Beitler-McKee Optical Co., Pittsburgh, Pa. Booth 103

The exhibit will feature the very latest styles of eyewear with display cabinets and trays so that they may be properly presented to the patient.

Featured among the frames will be the Golden 17. A display will also feature the latest in bifocals, as well as items of refracting equipment. William Driscoll, Earl Keller and E. F. Wildermuth will be in attendance.

Bell & Howell Co., Chicago, Ill. Booth 58

Bell & Howell Company will exhibit a complete line of motion picture equipment including cameras, projectors and editing equipment. The booth will feature the new amazing Model 202 magnetic sound recording Filmosound projector. With this new projector it is now possible for anyone to make his own sound motion pictures. All doctors are invited to attend the Bell & Howell exhibit booth for a demonstration.

N. P. Benson Optical Co., Minneapolis, Minn. Booths 84 & 85

Blakiston Co., New York, N. Y. Booth 83

The Blakiston Company exhibit features the sixth edition of a German classic —Meller's **Ophthalmic Surgery.** This practical and thorough volume contains descriptions of all up-to-date surgical procedures which have been thoroughly tried and proved to be safe. Each operation is discussed in such detail that it can be followed readily by anyone with a grasp of the fundamentals of ophthalmic surgery. Also on display is the third edition of Wolff's **Pathology of the Eye,** as well as many other popular practitioners' books in the field of ophthalmology.

Boll & Lewis Optical Co., Chicago, Ill. Booth 41

We are pleased to tell you that one of the main attractions for our booth at the coming Academy meeting will be that of featuring the Armorlite hard plastic prescription and industrial lenses.

These Armorlite lenses have stood the test for the past two and a half years and are the best plastic lenses known in the industry. We will have on display at that time, plastic bifocals in cataract form as well as all the various tints.

Plastic lenses are especially beneficial in cases of high myopia or hyperopia.

Burroughs Wellcome & Co., Inc., Tuckahoe, N. Y. Booth R-4

Featured will be:

AEROSPORIN® OTIC SOLUTION STERILE, which is bactericidal to gram-negative and gram-positive organisms, dehydrating and contains Aerosporin ® Sulfate Polymyxin B Sulfate in propylene glycol acidified with 1 per cent acetic acid.

POLYSPORIN® Polymyxin B — Bacitracin Ointment, Topical and Ophthalmic, which has a broad spectrum and is effective against styes, other eye infections, pyogenic skin infections and infections of wounds and burns.

Geo. H. Butterfield & Son, Portland, Ore. Booth R-9

We will exhibit Para-Curve corneal contact lenses together with supplies and equipment appertaining to the fitting thereof. We will also display our all-plastic artificial eyes.

Camel Cigarettes, New York, N. Y. Booths 27 & 28

CAMEL Cigarettes will mark your initials on an attractive plastic cigarette case filled with a package of those mild, flavorful CAMELS. This exhibit features a display of some of the tobaccos used in blending this famous cigarette which leads all other brands by many billions.

Cameron Surgical Specialty Co., Chicago, Ill. Booth 74

See the Anthony-Fisher Suction Coagulating Handle and Electrodes for nasal hemorrhage; cauteradios and cauterodynes for ocular, aural, nasal and oral electrosurgery, electrocauterization and electrocoagulation; telescopic bronchoscopes, esophagoscopes and laryngoscopes; the new Boros Flexible Esophagoscope and Flexible Gastroscopes with treated and coated lenses; electrodiagnostic lamps and instrument sets; mirror-type headlights; binocular spectacle loupes and other specialties for ophthalmology and otolaryngology.

Ciba Pharmaceutical Products, Inc., Summit, N. J. Booth R-10

Ciba Pharmaceutical Products, Inc., invites you to visit its exhibit which will feature PYRIBENZAMINE Hydrochlo-

ride, the antihistaminic which provides maximal allergic relief with minimal side effects.

Representatives in attendance will be glad to discuss the role of PYRIBENZAMINE in the treatment of various forms of allergy.

ClerSite Co., Chicago, Ill. Booth "C"

Coca-Cola Co., Chicago, Ill. Booth 26

Ice-Cold Coca-Cola served through the courtesy and cooperation of the Coca-Cola Bottling Company of Chicago, Inc., and the Coca-Cola Company.

Coreco Research Corp., New York, N. Y. Booth 59

Davis & Geck, Inc., Brooklyn, N. Y. Booth R-6

Davis & Geck, Inc., manufacturers of sutures and other surgical specialties, will exhibit the most comprehensive line of eye sutures available anywhere. These products have been developed in collaboration with outstanding ophthalmologists throughout the world. Both sutures and needles have been designed and perfected to meet the exacting conditions encountered in eye surgery. Exceptional sharpness of needle points will be demonstrated, with emphasis on the recently developed "inverted cutting edge" Atraumatic® needle. Of special interest will be Davis & Geck's new Aureomycin Dressing and Aureomycin Packing for various surgical procedures.

DeVilbiss Co., Toledo, Ohio Booths 15 & 16

The DeVilbiss Company, Atomizer Division, Somerset, Pennsylvania, welcomes your visit to its exhibit of professional and prescriptive types of Atomizers, Nebulizers and Vaporizers. A newly developed Respirator, No. 10, capable of screening out dust particles as small as 7/10 of one micron will be on display. Also on exhibit will be the No. 33 Pocket Atomizer, the No. 40 Nebulizer as well as the No. 149 Overnight Vaporizer.

Doho Chemical Corp., New York, N. Y. Booth 34

Doho Chemical Corporation is pleased to exhibit AURALGAN, the ear medication for the relief of pain in otitis media and removal of cerumen; RHINALGAN, the nasal decongestant which is free from systemic or circulatory effect and equally safe to use on infants as well as the aged; and the NEW OTOSMOSAN, the effective, nontoxic ear medication which is fungicidal and bactericidal (gram negative—gram positive) in the suppurative and aural dermatomycotic ears.

Encyclopaedia Britannica, Inc., Chicago, Ill. Booth R-7

Encyclopaedia Britannica announces the release of the 1952 edition, complete with new population figures based on the 1950 census.

This is the biggest revision of Encyclopaedia Britannica in a quarter of a century.

Britannica in several bindings, with yearbook and other services, will be available for inspection in our booth.

Grieshaber Swiss Eye Instrument, Chicago, Ill. Booth 38

We welcome your visit to our display of the inimitable Swiss ophthalmic cutting instruments made by Ernst Grieshaber of Switzerland.

Your attention is called to the ORIGINAL and GENUINE Grieshaber Eye Suture Needles with cutting edges. These needles are made to the highest standards of accuracy: each needle is an exemplification of perfection and is an instrument in itself.

On display also will be the Amsler Trocar Needle for anterior chamber, the finest Trephines for general eye surgery and corneal transplant as well as a carefully selected line of GRIESHABER Swiss Eye Knives.

As the EXCLUSIVE, Authorized Distributor in the United States and Canada for Ernst Grieshaber, Switzerland, we welcome your visit and offer prompt delivery from our stock of these very fine instruments.

Grune & Stratton, Inc., New York, N. Y. Booth 35

The Grune & Stratton booth will be the center of interest for those seeking new works in ophthalmology and otolaryngology. Books of importance which will be displayed for the first time at the convention include **Progress in Ophthalmology and Otolaryngology, Vol. I**, 1952, edited by Wiener, Maumenee, Ireland and

Sullivan; **Ophthalmic Plastic Surgery,** by Fox; **Surgery of the Eye,** third revised, reset and greatly enlarged edition, by Wiener and Scheie; **Physiology of the Eye, Vol. I, Optics** and **Vol. II, Vision,** by Linksz. In addition to these books, all published in the second half of 1952, Grune & Stratton will have on display a complete list of titles and journals in ophthalmology and otolaryngology.

Guild of Prescription Opticians of America, Inc., New York, N. Y.
Booths 43 & 44

Paul B. Hoeber, Inc., New York, N. Y. Medical Book Department of Harper & Brothers **Booth 32**

Be sure to visit the Hoeber-Harper booth where you will find and have the opportunity to examine the newly published Reese's **Tumors of the Eye;** Samuels & Fuchs' **Clinical Pathology of the Eye;** the complete 2-volume set of Berliner's **Biomicroscopy of the Eye;** the new work, Scott-Brown's **Ear, Nose & Throat;** Stallard's **Modern Practice in Ophthalmology;** Gesell's **Vision** and Ritvo's **Diseases of the Skull** as well as such books as Johnson's **Speech Handicapped School Children,** Wilton's **How to Overcome Stuttering** and many others. At booth 32 you will find the entire list of distinguished Hoeber books in print.

Hollister-Stier Laboratories, Wilkinsburg, Pa. **Booth 46**

The recognition of allergic symptoms is important in the practice of otolaryngology and the diagnosis and treatment is advanced through the Laboratories' services.

Hollister-Stier Laboratories are devoted exclusively to the preparation of allergenic materials and have worked with the American Society of Ophthalmologic and Otolaryngologic Allergy since its inception to prepare extracts which are useful for diagnosing and treating allergic patients.

Dry powders and liquid diagnostic extracts for pollens and other allergenic factors are displayed with therapeutic extracts and treatment dilutions. Many accessories for office and laboratory use are being demonstrated and literature on all items is available.

Our representatives will be glad to answer any inquiry or discuss any particular problem. Your visit is most welcome.

The House of Vision, Inc., Chicago, Ill.
Booths 60, 61 & 62

The House of Vision will have another unique exhibit of foreign ophthalmic equipment and many new devices which should prove very interesting to the medical profession.

The Haag-Streit instruments will be on display: the Goldmann Perimeter (with central scotoma attachment); the Pfluger Perimeter; the Goldmann Slit Lamp (with teaching eye-piece and tyndallimeter); and the Javal-Schiotz Ophthalmometer; the Amsler Ophthalmoscope.

There will be the Zeiss-Opton Slit Lamp; the Zeiss-Opton Refractionists' Set; and the Zeiss-Winkel Refractionometer; also the Aimark Perimeter and the Aimark Slit Lamp; different skiascopy racks; various muscle tests, trial frames and a new line of trial sets; Lensometers, lamps, magnets; Keeler and Hamblin diagnostic equipment, and a host of others.

On display will also be lenses that were designed and ground by The House of Vision in very unusual and difficult corrections, such as have been described in the "Scientific Corner" in the ophthalmologic journals.

Ion Optical Co., Detroit, Mich. Successors to Uhlemann Optical Co. of Michigan
Booth R-5

Bonded Featherweight Cataract Lenses; new type Bifocal and Trifocal; Jenconic Lenses for Anisometropic corrections.

Keeler Optical Products, Ltd., London, England **Booth 77**

Make a point of seeing these fine new instruments in the well-established Keeler range. They are all designed in collaboration with surgeons to give improved performance and encourage more modern technics.

PANTOSCOPE: A unique all-purpose mains ophthalmoscope with unsurpassable performance for direct or indirect examinations by polarized or natural light. There is also iris diaphragm control of illuminated area and a brilliant variable

slit for corneal and anterior chamber examinations. In addition the Pantoscope is ideal for red-free, focal or scleral transilluminations. With the speculum attachment it is a high intensity otoscope.

NEW "CONTINENTAL" OPHTHALMOSCOPE: Redesigned optics and improved styling make this new version of the popular "Continental" even more valuable for superb performance in direct or indirect ophthalmoscopy.

ASHER LAW STEREOSCOPE (Brit. J. Ophth. 36:225, 1952): This important new orthoptic instrument is proving a valuable aid to more accurate diagnosis and more satisfactory treatment. The Stereoscope obviates the disadvantages of conventional instruments with which convergence and accommodation can seldom be exercised in normal relation. The mechanics permit variable loci for the cards, thus ensuring that accommodation and convergence can really be controlled in normal or abnormal relation. The improved-design cards give maximal stimulus to fusion.

HALLPIKE EAR MICROSCOPE: Developed in collaboration with a Medical Research Council team, this portable instrument gives monocular vision with high magnification and intense 12-watt illumination which revolutionizes diagnostic and operative otology. Patented optics and silvered lamps give tremendous light gain. Shadowless deep penetration is obtained by actually looking through the light beam.

KEELER OPERATING SPECTACLE: Giving magnification X 2, working distance 25 cm, the galilean telescopes give a remarkable flat and colorfree 35 mm. field of view. Novel design allows vision to change from magnified image to wide-angle unmagnified operation field without change of focus or position. The unique "slip-on" telescope attachment ensures rapid manipulation. Both frame and telescope unit are individually fitted to eliminate adjustments and thereby ensure rigidity, comfort and best vision.

Keystone View Company, Meadville, Pa.
Booths 23 & 24

The Keystone View Company is exhibiting again a complete line of Orthoptic Training equipment. Keystone Prescription Services for home training will be featured. The Keystone Overhead Projector will be of interest to doctors who lecture or teach. It handles standard slides, 2" x 2" slides and microscopic slides. Competent technicians will be on hand to explain equipment and technics.

Kono Manufacturing Co., Woodside, N. Y.
Booth 76

The Kono Manufacturing Company offers an interesting and informative exhibit centered around their recently published booklet **Rare Visual Aids.** The result of intense ophthalmologic research and an accurate compilation of actual case histories, it represents the tremendous advance in the field of corrective frames necessitated by irregular and diseased physiognomonic structures. Kono demonstrates a practical solution for achieving custom fit by the use of molds which can be made easily and inexpensively by the ophthalmologist in his own office. In addition they are featuring a fine selection of frames for use in ordinary eye conditions, in a wide variety of styles, colors and materials.

Lea & Febiger, Philadelphia, Pa.
Booth 51

Lea & Febiger will exhibit many outstanding books, including Town—**Ophthalmology**; Spaeth—**Principles and Practice of Ophthalmic Surgery**; Krimsky—**Management of Binocular Imbalance**; Cowan—**Refraction of the Eye**; Gifford—**Handbook of Ocular Therapeutics**; Ballenger—**Diseases of the Nose, Throat and Ear**; Ballenger—**Manual of Otology, Rhinology and Laryngology**; and many others of special interest.

J. B. Lippincott Co., Philadelphia, Pa.
Booth R-8

J. B. Lippincott Company presents, for your approval, a display of professional books and journals geared to the latest and most important trends in current medicine and surgery. These publications, written and edited by men active in clinical fields and teaching, are a continuation of more than 100 years of traditionally significant publishing.

Mager & Gougelman, Inc., Chicago, Ill.
Booth 20 and ½ of 19

Mager & Gougelman's technical exhibit will consist of the finest display of artificial eyes in both glass and plastic. The display will include all types of mo-

tility implants, sphere implants, radiation therapy eye shields, intraocular foreign body locators, drains and conformers. The display will feature facial restorations and orbital appliances.

Maico Co., Inc., Minneapolis, Minn.
Booths 66 & 67

The Maico Company will exhibit an expanded line of clinical and professional audiometers, together with the Train-Ear for speech teaching and oral rehabilitation. Also to be shown are Model J and L hearing aids, both of which combine small size with fidelity of tone and range of amplification.

Mann Instrument Co., Oakland, Calif.
Booth R-17

Mann Instrument Company will display a new, modern line of ophthalmic instruments, including a new lensmeter, widely advertised Stanloscope, which is an orthoptic instrument utilizing polarized light and projecting two identical images upon a screen. This instrument is ideal for all types of fusion training. It is used in conjunction with moving pictures or still projection. George Young Threshold Tests will be displayed. This test is a screening test used as an aid in the diagnosis of glaucoma. Other instruments to be displayed will be the precise tangent screen, optical frame cabinet and dispensing set, consisting of fitting table, stool and chair. Mann Instrument Company will also display its new motorized perimeter.

Marcelle Cosmetics, Inc., Chicago, Ill.
Booth 54

New developments and new products in the Marcelle hypo-allergenic Cosmetic line will be demonstrated at the Marcelle exhibit. Representatives will include members of the Marcelle research organization who will give data and information on the role of Marcelle hypo-allergenic Cosmetics in the physician's practice.

Medical Case History Bureau, New York, N. Y. **Booth 112**

A handy, compact method for keeping complete case histories is the feature here. In the Info-Dex Record System, charts are so arranged on folders that subsequent cards may be attached by means of a cotter pin which holds the cards together in correct sequence. Different colored cards for various types of data such as operative reports, laboratory, or x-ray findings are obtainable. Included in this system is an automatic cross-indexing. Steel filing cabinets in which to house these history charts and bookkeeping records are also displayed. Representatives at the exhibit will gladly consult with you on any record problem or help you systematize your office routine.

Medical Film Guild, Ltd., New York, N. Y.
Booth "A"

"Medical Films that Teach" presents a refresher course in fundamental medical problems. Each film subject is produced in the manner of a textbook, profusely illustrated, offering information comparable to that found in postgraduate courses as presented at our leading medical schools. These films review such subjects as: (1) biomicroscopic examination of the eye; (2) allergy, immunology and treatment; (3) anatomy of the ear; (4) clinic on deafness; (5) chronic purulent otitis media; (6) otitis media in pediatrics; (7) clinic on acute mastoiditis; (8) function of the ear in health and disease; (9) inner ear; (10) clinic on sigmoid sinus thrombosis; (11) otoscopy in inflammation; (12) clinic on petrositis with meningitis and many others.

These films are available at no charge to intern groups, hospital staff conferences, limited nurses' groups and to general medical meetings. This includes projection service at no charge and is arranged through grants for postgraduate instruction.

Merck & Co., Inc., Rahway, N. J.
Booth 110

Merck & Co., Inc. is featuring CORTONE, HYDROCORTONE, NALLINE, and other medicinal preparations.

CORTONE has produced striking clinical improvement in rheumatoid arthritis and related rheumatic diseases; bronchial asthma; eye diseases including nonspecific iritis, iridocyclitis and uveitis; and skin diseases including cases secondary to drug reactions.

HYDROCORTONE is recommended for injection into the articular cavity of a rheumatoid or osteoarthritic joint.

NALLINE is a specific antidote in the treatment of overdosage with morphine and its derivatives, as well as meperidine and methadone.

Representatives at the Merck booth will be glad to provide information on these and other medicinal preparations such as antibiotics, NEO-ANTERGAN, URECHOLINE, and VINETHENE.

E. B. Meyrowitz Surgical Instruments Co., Inc., New York, N. Y.
Booths 79 & 80

Modern Optics, Inc., Houston, Texas
Booths 108 & 109

How high do you fit the seg? What is needed in the correct fitting of 3-way lenses will be demonstrated at the Modern Optics exhibit booth. This will include photographing the reading depth of each visitor at the exhibit

Of special interest to the ophthalmologists will be the experiences of the past year in prescribing the Modern Lenticular Cataract 3-way lens, which was first shown at the 1951 Academy.

Also on display will be a demonstration of threshold-welding, an exclusive Modern Optics manufacturing process.

C. V. Mosby Co., St. Louis, Mo.
Booth 86

A great deal of pertinent and timely information is on exhibit at the C. V. Mosby booth where you are invited to visit and browse at your leisure. Among the recent releases are **Volume 5**, Duke-Elder, **Textbook of Ophthalmology**; Scobee, **The Oculorotary Muscles**; Sorsby, **Systemic Ophthalmology**; Hansel, **Clinical Allergy**; Foldes-Weinstein, **The Glaucomas**; Pascal, **Visual Optics** and many others.

V. Mueller & Co., Chicago, Ill.
Booths 1 thru 6

V. Mueller & Company (Chicago) have prepared an unusually comprehensive exhibit of special and standard instruments, both domestic and imported, for all eye, ear, nose and throat surgery. A full line of superb instruments for eye work will be shown, as will a complete selection of instruments for plastic surgery, instruments—including the Shambaugh Microscope—for the fenestration procedure, modern equipment for suspension laryngoscopy, and many others.

New Era Optical Co., Chicago, Ill.
Booths R-19 & R-20

We will show the Maddox Cheiroscope for home training and office use; the New Era trial lens cabinet; the Omnitrainer, which is an instrument for all types of orthoptic training and diagnostic work; professional record materials, including cabinets and cards; the New Era ophthalmic hydraulic chair, and a general line of testing and diagnostic equipment.

New Jersey Optical Co., Irvington, N. J.
Booth 48

Mr. Sydney Freedman, vice president of the New Jersey Optical Company, reports that their new satin finish Heiress will be featured at the National Convention in Miami. He says that this Heiress frame, which is 1/10 12 K. G. F. throughout, has been most successful to date, in both plain and engraved.

Novocol Chemical Manufacturing Co., Inc., Brooklyn, N. Y. **Booth 68**

A cordial invitation is extended to all to visit with us. A qualified representative will be in attendance to explain the many advantages to be enjoyed with the use of MONOCAINE HYDROCHLORIDE solution (for local anesthesia) and the ANESTUBE SYSTEM. The growing popularity of MONOCAINE HYDROCHLORIDE solution and the ANESTUBE SYSTEM with ophthalmologists, otolaryngologists and plastic surgeons merits a few minutes of your time at booth 68.

Obrig Laboratories, Inc., New York, N. Y.
Booth 102

In addition to the exhibit on solutionless contact lenses and diagnostic lenses, the Obrig Laboratories, Inc. will display "PLAS-TITE," a soft plastic material which can be applied to nose bridges, plastic temples, and frames. It feels like soft rubber.

Paravox, Inc., Cleveland Ohio
Booth R-13

Exhibited by Paravox, Inc., is a complete line of fine hearing aids, and Photoscription, the new and efficient "prescription fitting" method of selecting hearing aids. For the first time Photoscription accurately shows by photograph the sound wave as it leaves the Paravox receiver, just as it would enter the ear. By keying the aid's performance to the

audiogram of the patient, much improved fitting is possible, and the aid's photograph serves as a perpetual record for future repair checks. Only Paravox has Photoscription, the new "prescription fitting."

Chas. Pfizer & Co., Inc., Brooklyn, N. Y. Booth R-14

Terramycin, newest of the broad-spectrum antibiotics, forms a dramatic central feature of the display of Chas. Pfizer & Co., Inc. The newest dosage forms of Terramycin are exhibited and indications for its use are described.

Philip Morris & Co., Ltd., Inc., New York, N. Y. Booth 75

Philip Morris and Company will show the results of research on the irritant effects of cigarette smoke. These results show conclusively that Philip Morris cigarettes are less irritating than other cigarettes. An interesting demonstration will be made on smokers at the exhibit which will show the difference in cigarettes.

George P. Pilling & Son Co., Philadelphia, Pa. Booths 63, 64 & 65

You are cordially invited to visit the Pilling exhibit booths to see and hear about the new Jackson System of Bronchoscopic Telescopes. These new telescopes will embody many interesting and new features for bronchotelescopy. Also on display will be a complete line of the Chevalier Jackson instruments for bronchoesophagology as well as a complete line of fine Pilling eye, ear, nose and throat instruments. Trained demonstrators will be glad to discuss all new and interesting features.

Precision-Cosmet Co., Inc., Minneapolis, Minn. Booth "B"

The focal point of this exhibit will be a series of life-size models depicting the surgical technic of the Allen Buried Muscle Cone Implant manufactured exclusively by Precision. Other Precision products on display will include the Lens Hardening Unit, Take-Off Chiller, Duffens Automatic Sphere Polisher, Contact Lenses, Plastic Artificial Eyes, Cencor 28m/m straight top Multifocal Lenses, Trial Frame for Small Children and the P.D. Lite & Meter.

Professional Press, Inc., Chicago, Ill. Booth 50

The Eye, Ear, Nose and Throat Monthly is in its thirty-first year of pulication. It has striven constantly to give the profession a monthly journal containing articles and other material of practical interest and value.

Radium Chemical Co., Inc., New York, N. Y. Booth 30

Radium Chemical Company, Inc., will display the Monel metal radium nasopharyngeal applicator, used for the treatment of hyperplastic lymphoid tissue at the orifice of the eustachian tube and the fossa of Rosenmuller. A complete line of instruments for the application of radon will also be shown.

Ritter Co., Inc., Rochester, N. Y. Booths 69 thru 73

On display will be complete treatment units and motor hydraulically operated chairs for eye, ear, nose and throat, a compact x-ray unit specifically designed for office use; an explosion-proof operating table for eye, ear, nose and throat surgery; automatic sterilizers, bone surgery engine, surgical cuspidors and mobile stools.

Ask about our professional office planning service.

W. B. Saunders Co., Philadelphia, Pa. Booth 49

Featured at the Saunders Exhibit will be the newly published **Atlas of Ophthalmic Pathology** (prepared under the joint sponsorship of the Academy and the Armed Forces Institute of Pathology).

Also on display will be such books as Berens' **The Eye and its Diseases**; Tait's **Textbook of Refraction**; Gifford-Adler's **Ophthalmology**; Jackson & Jackson's **Bronchoesophagology**; Ogle's **Researches in Binocular Vision**; and **Dorland Dictionary**; and many others.

Schering Corp., Bloomfield, N. J. Booth 33

Members of the American Academy of Ophthalmology and Otolaryngology and their guests are cordially invited to visit the Schering exhibit where new therapeutic developments will be featured.

Schering representatives will be present to welcome you and to discuss with you these products of our manufacture.

Selney Co., New York, N. Y. Booth R-1

You are cordially invited to visit our booth where we are featuring STERETAINER, a new disposable unit containing sterile solutions of Atropine, Pilocarpine, Homatropine, Eserine, Hyoscine, Tetracaine and Mydriphen.

The STERETAINER completely removes the danger of secondary, postoperative infections due to contaminated solutions.

The new mydriatic, MYDRIPHEN, clinically tested on premature infants, will also be featured in addition to sterile ophthalmic solutions for patients' prescription requirements.

Our Director of Research, Robert R. Feinstein, will be available for discussion of technical aspects relating to our products.

Shuron Optical Co., Inc., Geneva, N. Y. Booths 81 & 82

The Shuron Optical Company, Inc., will display a complete line of modern frames and mountings, Panorame vision lenses and the exclusive design spectacle cases. Featured will be the Browline frame family with all Zyltop rims and combination top rims of Zyl and gold filled in many colors for all members of the family.

Smith, Kline & French Laboratories, Philadelphia, Pa. Booth 45

We extend to you a cordial invitation to visit our booth where NEO-PENIL, a new chemical derivative of penicillin which produces higher concentration **in lung tissue** and **in sputum,** will be featured. Our representatives will be happy to discuss the encouraging prospect NEO-PENIL thus offers in the treatment of infections of the upper respiratory tract.

Soft-Lite Lens Co., Inc., New York, N. Y. Booth 104

The Soft-Lite booth for the Academy Meeting will feature a giant-size Comparator, which will demonstrate the difference between Soft-Lite and other absorptive lenses. In addition, Soft-Lite Lenses will be displayed in many of the forms available through Soft-Lite distributors. In attendance at the Soft-Lite booth will be Nat Singer, George F. Pearce, Al Kohler and Fred Slosson.

Sonotone Corp., Elmsford, N. Y. Booth 78

Sonotone unveils two new audiometers, one for general diagnostic, the other for advanced diagnostic and research applications. This new line for the first time offers full facilities to the American otologist for the Luscher Monaural Recruitment and the Denes and Naunton Difference Limen tests now coming into use on the Continent, plus the wide variety of other audiometric tests which are standard in American diagnostic procedures. Also included for the first time are facilities for a continuous check on calibration accuracy as to both frequency and output. Among other features are continuously variable frequency and attenuation, automatic pulsing or interruption, tone amplitude "wobbulation," a variety of masking tones including "white noise," calibrated control of masking-tone intensity, input connections for recorded and "live" speech with a panel meter for monitoring.

C. M. Sorensen Co., Woodside, L. I., N. Y. Booth 42

The C. M. Sorensen Company is exhibiting various suction and pressure units; ear, nose and throat treatment units; hydraulic chairs; central suction and pressure installations.

Storz Instrument Co., St. Louis, Mo. Booths 99, 100 & 101

Storz Instrument Company exhibit will display eye, ear, nose, throat and rhinoplasty instruments and equipment. Included will be a number of newer items of instruments and equipment, also a display of Zeiss-Opton products.

Sturgis-Grant Productions, Inc., New York, N. Y. Booth R-2

Specialized production of scientific teaching films in the fields of ophthalmology and otolaryngology is being demonstrated by the producers of the films **The Embryology of the Eye** and **Glaucoma.** The technics employed in producing both "live-action" and "animation" films are graphically illustrated by exhibit material, which also indicates the wide variety of projects to which these technics may be applied. The use of ster-

eoscopic slides in the visual presentation of medical subjects is also shown. Representatives at the exhibit invite discussion of film ideas and production problems.

Surgical Mechanical Research, Los Angeles, Calif. Booths 93 & 94

We will exhibit equipment advertised in this issue of the TRANSACTIONS, as well as our new electrically heated wash bottle, physician's grip and numerous small items for the office.

Thermosector Co., Oak Park, Ill. Booth 31

The Thermosector, an electrosurgical unit expressly designed for eye, ear, nose and throat technics in office and hospital, will be exhibited and demonstrated. You are invited to investigate the superior features of this device, which is preferred by leading hospitals, clinics and professional men.

Titmus Optical Co., Inc., Petersburg, Va. Booth 56

Highlighting the Titmus exhibit will be a representative display of 36 different lens styles in finished form except for edging. They will be arranged so they may beconveniently picked up for inspection of quality and design.

Lenses being shown nationally for the first time this year include the +0.50 and +2.50 Base Straight Tops for high minus Rx and the Contra-Glare Uniform Overlay in Straight Top Bifocals and Titmus "D" Bifocals. Infra-Bar and Blended Green Straight Tops will again be shown.

The firm's delegation will be headed by E. H. Titmus, Jr.

Travenol Laboratories, Inc. (Subsidiary of Baxter Laboratories, Inc.) Morton Grove, Ill. Booths R-21 & R-22

The new drug PIROMEN is a sterile, nonprotein, nonantigenic bacterial polysaccharide prepared as a colloidal dispersion of desacchromin for parenteral use. PIROMEN is a stimulant for the endocrine and reticuloendothelial systems, proven of value in the treatment of certain skin disorders, allergies, and ophthalmic diseases such as iridocyclitis, keratitis, uveitis, iritis and corneal ulcer.

Uhlemann Optical Co. of Illinois, Chicago, Ill. Booths 52 & 53

Uhlemann wishes to present to the members of the Academy a distinctively new bifocal lens, the Visaccon. This lens is designed to eliminate within .12 dp. the marginal astigmatism encountered when looking through the reading field of multifocal lenses.

In addition, we invite you to see our outstanding display of "glasses by Uhlemann" . . . a collection of styles with technical qualities.

Univis Lens Co., Dayton, Ohio Booths 39 & 40

Victory Optical Manufacturing Co., Newark, N. J. Booth 111

Visionez Corp., St. Cloud, Minn. Booth R-12

Dr. Max M. von Stirne, Philadelphia, Pa. Booth R-18

We manufacture and import surgical instruments for ophthalmologists, only.

Because we specialize, we should, and presume to, sell fine equipment and give excellent service to the profession.

Welch Allyn Co., Auburn, N. Y. Booth 17

White Haines Optical Co., Indianapolis, Ind. Booths 21 & 22

The White Haines Optical Company will exhibit in its booths at the 1952 meeting of the American Academy of Ophthalmology and Otolaryngology various ophthalmic products and ophthalmic equipment.

Williams & Wilkins Co., Baltimore, Md. Booth 36

The Williams & Wilkins Company booth will have on exhibit books covering all phases of problems of the eye, ear, nose and throat. Of special interest is the new fifth edition of Turner's **Diseases of the Nose, Throat and Ear,** edited by D. J. Guthrie. This ever-popular book has been out of print for some years. Its many friends will be glad to welcome the new, up-to-date version. See also Portmann's **Diseases of the Ear, Nose and Throat.** May's **Diseases of the Eye,** Van Alyea's **Nasal Sinuses,** and many other helpful books on diagnosis, treatment, surgery, and the basic sciences as they relate to this specialty.

Winthrop-Stearns, Inc., New York, N. Y.
Booth 25

The members of the American Academy of Ophthalmology and Otolaryngology are cordially invited to visit the Winthrop-Stearns booth. Featured products are OPH, decongestant, astringent, antiseptic eye drops; NTZ, a combination of the time-tested, dependable decongestant Neo-Synephrine, with the potent, well-tolerated antihistaminics Thenfadil and Zephiran; Neo-Synephrine, special ophthalmic preparation for mydriasis of short duration; Pontocaine, reliable topical anesthetic with prolonged action and good penetration.

Carl Zeiss, Inc., New York, N. Y.
Booth R-16

At the Academy Meeting there will be shown, for the first time, at the booth of Carl Zeiss, Inc., the new Zeiss-Opton Twin Lamp.

This is an operating lamp especially suitable for ear and eye surgery. The advantage of this lamp lies in its small, sharply defined field of illumination. The smallest area that can be illuminated is two inches. By a single movement both focusing and coincidence of the two fields of illumination can be set. A particularly high illumination is provided by the double light source. A high degree of security is assured inasmuch as no interruption in the examination and operation is caused by the failure of a lamp and the necessary replacement. Also new is the attachment for the Zeiss-Opton Slit Lamp Apparatus for gonioscopic work. In addition, the well-known Zeiss-Opton Slit Lamp Apparatus and Zeiss-Winkel Vertex Refractionometer will be on display. Also exhibited will be the Zeiss-Opton Stereo-Microscope, Hand Ophthalmoscope, Hand Slit Lamp and Diascleral Lamp.

Zenith Radio Corp., Chicago, Ill.
Booth 55

The Zenith Radio Corporation will exhibit their new air and bone conduction hearing aids, featuring an exclusive new heat and humidity resistant ceramic Permaphone and ingenious Reserve "A" Battery Switch. Both advances eliminate the prime causes of failure in an electrical hearing device. Zenith representatives will be pleased to explain how any physician can test one of these instruments for thirty days without cost or obligation.

Zylite Products Co., Inc., New York, N. Y.
Booth R-11

Zylite, first in frame fashion, will exhibit their latest "Zylite Originals." They will show the latest in frame color, style and construction, providing the refractionist with a complete frame service.

Zylo Ware Corp., Long Island City, N. Y.
Booth R-15

Zylo Ware Corporation will as usual have a complete display of ophthalmic frames in zylonite. Featured will be the Magic Empress and Magic Saucy frames, highly styled frames for the feminine taste, and the Monte Carlo frame and the Casa Blanca, both frames reflecting the trend to the heavier weight frame now being favored by men.

SECTION ON INSTRUCTION

FIFTY-SEVENTH ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

October 12-17, 1952
PALMER HOUSE
Chicago, Illinois

A. D. RUEDEMANN, M.D.
Secretary for Instruction — Ophthalmology

DEAN M. LIERLE, M.D.
Secretary for Instruction — Otolaryngology and Maxillofacial Surgery

The following condensed schedules of instruction courses are reprinted for the convenience of Fellows who have not yet placed orders for tickets.

Courses already sold out are so designated on the charts. Please do not request tickets for these courses.

For complete listing of course titles, instructors, and descriptive paragraphs about each subject, please refer to the March-April issue of the TRANSACTIONS.

General Instructions

Only members who have paid their 1952 dues may order instruction tickets in advance of the meeting. Members should order tickets only for their own use. Candidates for fellowship in the Academy are not privileged to reserve tickets in advance. After the session opens, no distinction between members, candidates and guests will be made in selling tickets.

Each period (1 hour) of instruction costs $2.00. *Orders unaccompanied by properly executed checks will not be filled.* Checks should be made payable to W. L. Benedict, M.D.

In case of inability to attend the meeting after order has been filled, a refund will be granted if requested prior to October 10.

If courses requested are not available and it is impossible to substitute, a refund will be granted. Refunds will not be sent by mail. A blue refund card will be placed in the Advance Registration Envelope which is received at the time of registration. This should be presented at the Instruction Desk for payment.

All orders for tickets will be filled according to postmark on envelope containing check. Telegrams will not be honored. Previous to October 1, send orders to 100 First Avenue Building, Rochester, Minnesota; after October 1, mail orders direct to W. L. Benedict, M.D., c/o Palmer House, Chicago, Illinois. Advance orders will not be filled if received after October 9.

Ordering Instruction Tickets

An *individual* course is complete in one (1) period. Most individual courses are repeated on two or three different days allowing the subscriber to choose the day on which he wishes to attend.

A *continuous* course lasts two (2) or more periods as indicated. It is not possible to purchase a ticket to only *part* of

a continuous course.

At the end of this section is printed a colored sheet to be used when ordering tickets in advance. Specify preferences on this sheet and mail to the executive office. Careful attention to instructions will ensure the correct filling of your requests. *Since number of tickets for each course is limited, be sure to list second and third preferences for each period in case first preference is already sold out. In every case, effort will be made to furnish first choices.*

If you are requesting registration in the continuous courses, please specify preferences for individual courses as substitutes in order to avoid disappointment should the continuous courses which you select be sold out when your order is received.

Acknowledgment of Advance Order

As soon as an advance order is filled, a postcard, listing the courses that have been reserved for him, will be mailed to the subscriber. Please do not ask to change courses after your order has been filled. The postcard is merely an assurance that your order has been filled; it is not necessary for registration at the Academy meeting.

Claiming Instruction Tickets

No tickets will be sent through the mails. Members who have ordered instruction course tickets will register at the *Advance Order Section* of the Registration Desk during the week of the Convention to secure registration badge, tickets, and all convention material.

KEY TO DESIGNATION OF PERIODS

The periods will be designated with the following key: Select tickets for—

Otolaryngology, forenoons, periods 1-2-3—Ophthalmology, afternoons, periods 4-5-6

Day	(M)onday	(T)uesday	(W)ednesday	(Th)ursday
Period a.m.		1 2 3	1 2 3	1 2 3
Period p.m.	4 5 6	4 5 6	4 5 6	4 5 6

M-4 ..... 2:00 p.m. to 3:00 p.m.
M-5 ..... 3:15 p.m. to 4:15 p.m.
M-6 ..... 4:30 p.m. to 5:30 p.m.
T-1 ..... 9:00 a.m. to 10:00 a.m.
T-2 .....10:15 a.m. to 11:15 a.m.
T-3 .....11:30 a.m. to 12:30 p.m.
T-4 ..... 2:00 p.m. to 3:00 p.m.
T-5 ..... 3:15 p.m. to 4:15 p.m.
T-6 ..... 4:30 p.m. to 5:30 p.m.
W-1 ..... 9:00 a.m. to 10:00 a.m.
W-2 .....10:15 a.m. to 11:15 a.m.
W-3 .....11:30 a.m. to 12:30 p.m.
W-4 ..... 2:00 p.m. to 3:00 p.m.
W-5 ..... 3:15 p.m. to 4:15 p.m.
W-6 ..... 4:30 p.m. to 5:30 p.m.
Th-1 ..... 9:00 a.m. to 10:00 a.m.
Th-2 .....10:15 a.m. to 11:15 a.m.
Th-3 .....11:30 a.m. to 12:30 p.m.
Th-4 ..... 2:00 p.m. to 3:00 p.m.
Th-5 ..... 3:15 p.m. to 4:15 p.m.
Th-6 ..... 4:30 p.m. to 5:30 p.m.

(Familiarity with this key will be helpful and will avoid confusion)

CHANGES IN INSTRUCTION COURSE PROGRAM AS LISTED IN THE MARCH-APRIL 1952 TRANSACTIONS

Dr. Wilfred E. Fry of Philadelphia will give the lecture on "Glaucoma Secondary to Uveitis" on W-5 in Course 33, replacing Dr. F. Bruce Fralick.

Dr. E. King Gill will conduct Course 426, "External Otitis." Dr. William D. Gill, deceased, was originally scheduled to deliver this lecture.

Because of the death of Dr. Richard G. Scobee continuous course 42 has been cancelled.

The title of Course 445 given by Dr. Leland G. Hunnicutt on T-1 and Th-1 has been changed from "When and How to Make Tissue Examination: A Practical Office Concept" to "Control of Hemorrhage in Ear, Nose and Throat."

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
1	Allen, J. H., et al	Bacteriology & Infectious Diseases	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
2	Ascher	Aqueous Veins in Glaucoma	2 PERIODS W-5, W-6
3	Bedell	Medical Ophthalmoscopy	4 PERIODS M-5, T-5, W-5, Th-5
4	Berliner	..... SOLD OUT	
5	Birge, et al	New Theories — Retinal Venous Thrombosis	2 PERIODS Th-4, Th-5
6 or 7	Boeder	Aniseikonia & Space Eikonometry	2 PERIODS W-4, W-5 or Th-4, Th-5
8	Brown, et al	Eye Muscles	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
9	Clark	Surgery to Ocular Muscles	2 PERIODS M-5, M-6
10 or 11	Copeland	Streak Retinoscopy	4 PERIODS M-4, M-5, T-4, T-5 or W-4, W-5, Th-4, Th-5
12	Costenbader, et al	Concomitant Strabismus	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
13	DeVoe	Clinical Application of Gonioscopy	2 PERIODS Th-5, Th-6
14	Elwyn	Heredodegenerative Diseases of Retina	2 PERIODS M-5, M-6
15	Fink	..... SOLD OUT	
16	Hardy, et al	Basic Principles of Orthoptics	4 PERIODS M-4, T-4, W-4, Th-4
17	Hastings	Psychosomatic Medicine	2 PERIODS W-5, W-6
18 or 19	Henderson	Perimetry	2 PERIODS M-4, M-5 or Th-4, Th-5
20	Hughes, et al	Plastic Surgery	10 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, Th-4, Th-5
21	Jardon	Fitting of Ocular Prostheses	2 PERIODS W-5, W-6
22 or 23	Johnson, L. V.	Aqueous Tonography	2 PERIODS W-5, W-6 or Th-4, Th-5
24 or 25	Johnson, V. C., & Davies	Diagnostic Value of X-ray	2 PERIODS M-5, M-6 or T-5, T-6
26	Knighton, et al	Highlights of Glaucoma	8 PERIODS M-5, M-6, T-5, T-6, W-5, W-6, Th-5, Th-6
27	Kuhn	Industrial Ophthalmology	3 PERIODS M-4, T-4, W-4

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
28	Lebensohn, et al	Refraction	6 PERIODS W-4, W-5, W-6, Th-4, Th-5, Th-6
29	Lowrey	Military Ophthalmology	2 PERIODS W-5, W-6
30	Lyle	Neuro-Ophthalmology	4 PERIODS M-4, T-4, W-4, Th-4
31	McLean, et al	..... SOLD OUT	
32	Meek	Surgical Anatomy of Orbit	2 PERIODS T-5, T-6
33	Meyer, et al	Glaucoma	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
34	Ogle	Essential Aspects of Binocular Vision	2 PERIODS M-5, M-6
35	O'Rourke	Manifest & Cycloplegic Refraction	2 PERIODS T-5, T-6
36	Paton	Corneal Surgery	3 PERIODS T-4, W-4, Th-4
37	B. Payne, et al	Practical Eye Pathology	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
38	Pino	Bifocal Lenses & Presbyopia	2 PERIODS M-5, M-6
39	Romaine, et al	Ocular Motility	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
40 or 41	Schepens	Ophthalmoscopy in Diagnosis of Retinal Detachment	2 PERIODS M-5, M-6 or T-5, T-6
42	Scobee, et al	..... CANCELLED	
43	Stow, et al	Applied Clinical Pathology	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
44	Theobald, et al	Histopathology	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
45	Thorpe, et al	Slit Lamp Biomicroscopy	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
46	Vail & Minsky	Zonular Membrane & Cataract Surgery	2 PERIODS M-5, M-6
47	Wheeler & Knapp	Concomitant Strabismus	6 PERIODS M-5, M-6, T-5, T-6, W-5, W-6
48	Wiener	..... SOLD OUT	
49	Friedenwald	Measurement of Aqueous Flow	2 PERIODS W-4, W-5

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Monday

Key M-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
204	Atkinson	..... SOLD OUT .....
205	Baird	Acute Lesions of Cornea
206	Baker	Neuralgias of Head
212	Byrnes	Adjuncts in Teaching Ophthalmology
219	Cordes	Surgery of Congenital Cataracts
226	Falls	Ocular Geriatrics
235	Gipner	Recent Advances
245	Hogan	Pathology of the Choroid
256	Lloyd	Macular Lesions
262	McCaslin	Ocular Anesthesia
268	Osher	Diagnostic Approach to Squint Surgery
269	Owens & Owens	Retrolental Fibroplasia
272	Pfeiffer	Diagnosis with Aid of X-ray
274	Post, L. T.	Difficult Problems in Refraction
275	Post, M. H.	Operating Room Technic
280	Rychener	Lesions of the Lids
285	Spaeth	Lacrimal Gland & Sac
297	Whalman	Minor Surgical Procedures of Eye
A	Home Study	Embryology, Anatomy & Neuro-Anatomy

Key M-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
207	Barkan	Congenital Glaucoma
209	Berens	Uveitis: Etiology & Treatment
220	Cusick	Ptosis
224	Dunnington	Ocular Wound Healing
229	Fread	Ideal Refraction Equipment
252	Leinfelder	Papilledema & Optic Neuritis
255	Linksz	Visual Physiology
266	Newell	Rabbit Surgery
278	Rudolph	Advantages of General Anesthesia
290	Swan	Value of New Drugs
294	Veasey	Refraction Difficulties
B	Home Study	Physiology

Key M-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
201	Alvaro	Uveitis: Etiology & Treatment
203	Amenabar	Detachment of Retina: Treatment
216	Chace	Ocular Malingering
222	Davies	Clinical Pathology in Optic Atrophy
223	Dunlap	Divergence Anomalies
231	Fry	Differential Diagnosis of Lesions at Posterior Pole of Eye
233	Gemeroy	External Diseases of Eye
239	Gray	Intraocular Foreign Bodies
243	Hanser	Ocular Allergy
253	Lemoine	Vitreous Opacities
254	Lepard	Eye Diseases Common to Children
261	Marshall	Lid Infections
270	Payne, F.	Unilateral Visual Failure
277	Reese	Cataract Extraction
279	Rundles	Streak Retinoscopy
281	Scheie	Surgery in Congenital Glaucoma
C	Home Study	Optics & Principles of Biomicroscopy

SECTION ON INSTRUCTION

ADVANCE ORDER SHEET

All courses cost $2.00 for each period (one hour). Follow instructions explicitly and check for errors in listing before mailing.

Previous to OCTOBER 1

Send order to . . .

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
100 First Avenue Building, Rochester, Minnesota

After OCTOBER 1, mail to . . .

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
c/o Palmer House, Chicago, Illinois

Academy Candidates and Guests May Purchase Tickets at Registration Desk Only

(Over)

ADVANCE ORDER SHEET

ORDERS FILLED ONLY IF CURRENT DUES ARE PAID

NO REFUNDS WILL BE MADE AFTER OCTOBER 10

(Type or Print) Last Name First Name Middle Initial

Office Address

City Zone State

Days to be Present

☐ Monday

☐ Tuesday

☐ Wednesday

☐ Thursday

Total Amount Enclosed $_____

SELECTION OF COURSES ON REVERSE SIDE OF THIS BLANK

RECAPITULATION OF GENERAL INSTRUCTIONS

1. Monday, Tuesday, Wednesday, and Thursday are represented by the letters M, T, W, Th. The first period in the morning is represented by figure 1, second period by figure 2 and third period by figure 3. The first period in the afternoon is represented by figure 4, second period by figure 5, and third period by figure 6. All courses in otolaryngology and maxillofacial surgery are presented in the mornings. All courses in ophthalmology are presented in the afternoons.
2. Reservation of instruction tickets in advance of the meeting will be made ONLY for MEMBERS whose 1952 dues have been paid. Academy candidates and guests may make selection of courses ONLY at the General Registration Desk in Chicago.
3. Make check payable to W. L. Benedict, M.D.
4. Mail order promptly to ensure reservation of first choices. If necessary, an attempt will be made to rearrange schedule to include "first choices."

(Over)

SELECTION OF COURSES

PERIOD	FIRST CHOICE		SECOND CHOICE		THIRD CHOICE	
	Course No.	Lecturer	Course No.	Lecturer	Course No.	Lecturer
MON. 4						
P.M. 5						
6						
TUES. 1						
A.M. 2						
3						
P.M. 4						
5						
6						
WED. 1						
A.M. 2						
3						
P.M. 4						
5						
6						
THURS. 1						
A.M. 2						
3						
P.M. 4						
5						
6						

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Tuesday

Key T-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
202	Alvis	End Results of Cataract Wound Closure
204	Atkinson	Measures in Cataract Extraction
205	Baird	Acute Lesions of Cornea
206	Baker	Neuralgias of Head
217	Chandler	Secondary Glaucoma in Aphakia
228	Fralick	External Ocular Manifestations of General Disease
235	Gipner	Recent Advances
240	Guibor	Physiology of Ocular Muscles
256	Lloyd	Macular Lesions
257	Lueck	Practical Optics
262	McCaslin	Ocular Anesthesia
269	Owens & Owens	Retrolental Fibroplasia
272	Pfeiffer	Diagnosis with Aid of X-ray
275	Post, M. H.	Operating Room Technic
283	Siniscal	Chronic Lid Conditions
286	Spaeth	Plastic Surgery
297	Whalman	..... SOLD OUT .....
D	Home Study	Refraction

Key T-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
209	Berens	Uveitis: Etiology & Treatment
210	Berke	Surgery of Oblique Muscles
220	Cusick	Ptosis
224	Dunnington	Ocular Wound Healing
227	Falls	Practical Application of Heredity
229	Fread	Ideal Refraction Equipment
252	Leinfelder	Papilledema & Optic Neuritis
255	Linksz	Visual Physiology
276	Randolph	Handling Patients with Advanced Glaucoma
282	Simpson	..... SOLD OUT .....
291	Tait	Methods in Correction of Accommodation & Convergence Abnormalities
294	Veasey	Refraction Difficulties
296	Walsh	Interesting Neuro-Ophthalmologic Cases
E	Home Study	External Diseases, Bacteriology & Virology

Key T-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
201	Alvaro	Uveitis: Etiology & Treatment
203	Amenabar	Detachment of Retina: Treatment
216	Chace	Ocular Malingering
222	Davies	Clinical Pathology in Optic Atrophy
223	Dunlap	Divergence Anomalies
231	Fry	Differential Diagnosis of Lesions at Posterior Pole of Eye
233	Gemeroy	External Diseases of Eye
239	Gray	Intraocular Foreign Bodies
243	Hanser	Ocular Allergy
246	Iliff	Beta Irradiation Therapy
253	Lemoine	Vitreous Opacities
261	Marshall	Lid Infections
270	Payne, F.	Unilateral Visual Failure
273	Pischel	Retinal Detachment Surgery: Complications
279	Rundles	Streak Retinoscopy
281	Scheie	Surgery in Congenital Glaucoma
287	Steffensen	ACTH & Cortisone
F	Home Study	Perimetry & Neuro-Ophthalmology

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Wednesday

Key W-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
202	Alvis	End Results of Cataract Wound Closure
211	Bettman	Value of Goniotomies
213	Calhoun	Dystrophies & Degenerations of Cornea
217	Chandler	..... SOLD OUT .....
221	Cutler	Procedures Used in Enucleation
238	Gordon	Anatomy of Orbital Fascia
241	Guibor	Neuromotor Defects of Ocular Muscles
244	Harrington	Psychosomatic Phases
247	Jobe	Practical Physiologic Optics
263	McGuire	Heterophoria & Heterotropia
271	Perera	Retinal Detachment
283	Siniscal	Chronic Lid Conditions
284	Smith	Practical Points in Office Treatment
292	Theodore	Ocular Therapeutics
298	Wiener	Minor Lid Surgery
G	Home Study	Extraocular Muscles

Key W-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
208	Bellows	Changes in Crystalline Lens
210	Berke	Surgery of Oblique Muscles
215	Cassady	Dacryocystitis of Infancy
232	Gailey	Complications of Cataract Surgery
234	Gifford	..... SOLD OUT .....
237	Goar	Dystrophies of Cornea
249	Klien	Clinical Pathology of Retinal Lesions
264	Morrison	Office Management of Glaucoma
267	Olson	Iritis, Iridocyclitis, & Keratitis
291	Tait	Methods in Correction of Accommodation & Convergence Abnormalities
293	Trueman	Cerebral Lesions
H	Home Study	Ophthalmoscopy & Medical Ophthalmology

Key W-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
214	Callahan	..... SOLD OUT .....
218	Cholden	Informing Patient of Blindness: Psychiatric Aspects
225	Egan	Cross Cylinder Application & Theory
236	Givner	Systemic Disease
246	Iliff	Beta Irradiation Therapy
248	Katz	Corneal Dystrophies
250	Krewson	Clinical Significance of Oblique Muscles
251	Kronfeld	End Results in Glaucoma Surgery
254	Lepard	Eye Diseases Common to Children
258	Macnie	Head Trauma with Ocular Significance
259	Mangold	Proper Fitting of Glasses
260	Mann, M.	Hypertensive Changes in Fundus
265	Mullen	Ophthalmic Emergencies
287	Steffensen	ACTH & Cortisone
288	Stimson	Lenses for the Aphakic
299	Mann, M.	Refraction and Lens for Cataract Patient
I	Home Study	Pathology

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Thursday

Key Th-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
211	Bettman	Value of Goniotomies
213	Calhoun	Dystrophies & Degenerations of Cornea
221	Cutler	Procedures Used in Enucleation
238	Gordon	Anatomy of Orbital Fascia
242	Guibor	Neurology of Ocular Muscles
244	Harrington	Psychosomatic Phases
247	Jobe	Practical Physiologic Optics
263	McGuire	Heterophoria & Heterotropia
267	Olson	Iritis, Iridocyclitis, & Keratitis
271	Perera	Retinal Detachment
284	Smith	Practical Points in Office Treatment
292	Theodore	..... SOLD OUT .....
J	Home Study	Pharmacology & Therapeutics

Key Th-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
208	Bellows	Changes in Crystalline Lens
215	Cassady	Dacryocystitis of Infancy
232	Gailey	Complications of Cataract Surgery
234	Gifford	Dacryocystorhinostomy
249	Klien	Clinical Pathology of Retinal Lesions
258	Macnie	Head Trauma with Ocular Significance
264	Morrison	Office Management of Glaucoma
293	Trueman	Cerebral Lesions

Key Th-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
214	Callahan	..... SOLD OUT .....
225	Egan	Cross Cylinder Application & Theory
236	Givner	Systemic Disease
248	Katz	Corneal Dystrophies
250	Krewson	Clinical Significance of Oblique Muscles
259	Mangold	Proper Fitting of Glasses
260	Mann, M.	Hypertensive Changes in Fundus
265	Mullen	Ophthalmic Emergencies
287	Steffensen	ACTH & Cortisone
288	Stimson	Lenses for the Aphakic
299	Mann, W.	Refraction and Lens for Cataract Patients

CONTINUOUS COURSES — OTOLARYNGOLOGY

COURSE	SPEAKERS	TITLE	PERIODS USED
101	Altmann & Waltner	Histopathology of Ear—Part I	2 PERIODS T-1, T-2
102	Altmann & Waltner	Histopathology of Ear—Part II	2 PERIODS W-1, W-2
103 or 104	Bryan & Bryan	Cytologic Pathology of ENT Diseases	2 PERIODS T-1, T-2 or W-1, W-2
105 or 106	Conley	Cancer of Head & Neck	2 PERIODS T-1, T-2 or Th-2, Th-3
107	Converse	Nasal Plastic Surgery (Basic)	2 PERIODS W-1, W-2
108	Converse	Nasal Plastic Surgery (Advanced)	2 PERIODS Th-2, Th-3
109 or 110	Figi	Lesions of Mouth	2 PERIODS T-2, T-3 or W-2, W-3
111	Fowler & Montreuil	Histology of Ear — Vertical Sections	2 PERIODS T-1, T-2
112	Fowler & Montreuil	Histology of Ear — Horizontal Sections	2 PERIODS Th-1, Th-2
113 or 114	Hansel	Allergy	2 PERIODS T-1, T-2 or Th-1, Th-2
115	Jackson	Technic in Peroral Endoscopy	3 PERIODS T-3, W-3, Th-2
116	Kazanjian, et al	Traumatic Injuries of Face	2 PERIODS T-1, T-2
117 or 118 or 119	Lempert	Evaluating Degree of Hearing Function	2 PERIODS T-2, T-3 or W-2, W-3 or Th-2, Th-3
120 or 121	Lindsay	Vertigo	2 PERIODS W-2, W-3 or Th-2, Th-3
122 or 123	Maliniac	Maxillofacial Plastic Surgery	2 PERIODS T-1, T-2 or W-2, W-3
124	Marcus, et al	Hearing Problems in Children	2 PERIODS W-1, W-2
125	Moses	Speech & Voice Therapy	2 PERIODS W-2, W-3
126 or 127	Mueller	Practical Audiometry	2 PERIODS W-2, W-3 or Th-1, Th-2
128 or 129	Orton	Applied Surgery of Neck	2 PERIODS T-2, T-3 or Th-2, Th-3
130 or 131	Sellers	Equilibration	2 PERIODS T-1, T-2 or Th-2, Th-3
132 or 133	Semenov	Therapy in Pathology of ENT	2 PERIODS T-2, T-3 or Th-2, Th-3
134	Tucker, et al	Esophageal Diseases & Foreign Bodies	3 PERIODS T-1, W-3, Th-1
135	Tucker, et al	Laryngeal Surgery	3 PERIODS T-3, W-2, Th-2
136 or 137	Turchik	Nasal Obstruction	2 PERIODS W-1, W-2 or Th-2, Th-3

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Tuesday

Key T-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
401	Alexander	Adenoid Operation: Relation to Auditory Apparatus
403	Baron	Modified Radical Mastoidectomy
406	Boyden	Chronic Frontal Sinus Surgery
414	Craft	Nasal Allergy
417	Day	What the Otologist Can Do for Deafness
421	Fabricant	Modern Medication of Nose & Sinuses
426	Gill	External Otitis
427	Glick	Surgical Approaches on Neck
429	Goldman	Rhinologic Diagnosis & Treatment
439	Holinger	Esophageal Diseases in Infants & Children
441	Hosler	Cardiac Arrest During Operation
442	House	Surgery of Chronically Discharging Ear
445	Hunnicutt	Control of Hemorrhage
451	Kemler	Mirror Laryngology
460	Maxwell	Tumors of Face & Neck in Infancy & Childhood
462	McLaurin	Otitis Externa: Therapy
475	Reger	Auditory Malingering
477	Richards	Suggestions Concerning Office Practice
482	Seltzer	Plastic Surgery of Nose & Face-Lift Operation
485	Shea	Intranasal Surgery
491	Tremble	Nose & Throat Medication
497	Wilkerson	Hearing & Speech Center for Your City

Key T-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
402	Ashley	Dust Therapy in Respiratory Allergy
404	Becker	Plastic Surgery of Protruding Ears
407	Canfield	Audiology for Practicing Otologist
415	Croushore	Hemorrhage: Prevention & Control
422	Farrior & Bagby	Surgical Anatomy of Ear
425	Friedberg	Diseases of Larynx
431	Hallberg	Nosebleed & Its Management
444	Hunnicutt	Chronically Congested Nose
453	Kirchner	Difficulty in Swallowing
464	Miller	Laryngeal Emergencies in Infants & Children
468	Ogura	Laryngectomy & Neck Dissection
470	Perlman	Tonsil & Adenoid Operation
471	Perlman	Serous Otitis
474	Putney	Surgery of Neck Tumors
480	Ryan	Headache
487	Simmons	Intranasal Dacryocystorhinostomy
489	Singleton	Tonsil & Adenoid Problem in Children
493	Van Alyea	Embryology of ENT
496	Walsh	Otitis Media: Treatment
501	Panel	Otological Problems

Key T-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
405	Bellucci	..... SOLD OUT .....
409	Cohen	Deviated Noses & Their Correction
411	Conley	Pigmented Mole & Malignant Melanoma: Treatment
412	Costen	Mandibular Joint Syndrome
416	Cullen	Anesthesia
423	Farrior & Carron	Fenestration Operation
424	Fisher	Esophageal Obstruction
428	Glorig	Evaluation of Methods for Measuring Auditory Acuity
430	Goodhill	..... SOLD OUT .....
432	Harrill	Bronchography
433	Hilger	Therapeutic Approaches to Inner Ear
438	Holinger	Pediatric Laryngology
443	Howard	Office Procedures in ENT
448	Juers	Hearing Tests
449	Keaster	Speech & Hearing in Children Under Five
458	Lewin	Facial & Nasal Reconstructive Surgery
459	Long	Chemotherapeutic & Antibiotic Agents
466	Morrison	Technic of Tonsillectomy
469	Pastore	A Lump in the Throat
473	Pressman	Rhinologist & Problem of Rhinoplasty
481	Sataloff	Treatment of Nerve Deafness
484	Shambaugh	Failures in Fenestration Surgery
490	Thornell	Intralaryngeal Arytenoidectomy
494	Van Alyea	Chronic Sinusitis

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Wednesday

Key W-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
401	Alexander	Adenoid Operation: Relation to Auditory Apparatus
403	Baron	Modified Radical Mastoidectomy
408	Carhart	Audiogram in Otologic Diagnosis
413	Cottle	Septal Reconstruction Surgery
414	Craft	Nasal Allergy
417	Day	What the Otologist Can Do for Deafness
422	Farrior & Bagby	Surgical Anatomy of Ear
426	Gill	External Otitis
427	Glick	Surgical Approaches on Neck
429	Goldman	Rhinologic Diagnosis & Treatment
440	Holmes	Scars of Face
441	Hosler	Cardiac Arrest During Operation
450	Kelemen	Mechanical Injuries of Hearing Organ
451	Kemler	Mirror Laryngology
456	LeJeune	Technic of Intralaryngeal Surgery
460	Maxwell	Tumors of Face & Neck in Infancy & Childhood
461	McConnell	Speech Audiometry for Otologist
462	McLaurin	Otitis Externa: Therapy
463	McNally	Labyrinthine Tests
467	Neivert	Care of Instruments
476	Reger	Practical Office Audiometry
477	Richards	Suggestions Concerning Office Practice
479	Roberts, S. E	Head & Neck Syndrome
483	Senturia	External Otitis
495	Von Leden	Tracheotomy
500	Panel	Tumors & Infections of Neck
503	Panel	Rhinology

Key W-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
404	Becker	Plastic Surgery of Protruding Ears
407	Canfield	Audiology for Practicing Otologist
415	Croushore	Hemorrhage: Prevention & Control
420	Erich	Fractures of Facial Bones
431	Hallberg	Nosebleed & Its Management
433	Hilger	Therapeutic Approaches to Inner Ear
435	Hill	Antro-Alveolar Fistulas
444	Hunnicutt	Chronically Congested Nose
447	Jordan	Otologic Conditions Complicated by Allergy
452	Kinney	Acoustic Trauma
455	Kuhn	Allergic Diseases of Ear
457	Lenz	Cancer of Nose & Throat: Radiotherapy
466	Morrison	Technic of Tonsillectomy
468	Ogura	Laryngectomy & Neck Dissection
470	Pearlman	Tonsil & Adenoid Operation
472	Pohlman	Plastic Ear Drum
488	Simonton	Recruitment of Loudness & Diplacusis Binauralis
490	Thornell	Intralaryngeal Arytenoidectomy
493	Van Alyea	Embryology of ENT
499	Panel	Diseases of Larynx, Neck, Tracheobronchial Tree & Esophagus

Key W-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
405	Bellucci	Submucous Resection of Nasal Septum
409	Cohen	Deviated Noses & Their Correction
418	Derlacki	Allergy of Ear
419	DeWeese	The Dizzy Patient
424	Fisher	Esophageal Obstruction
425	Friedberg	Diseases of Larynx
437	Hitschler	Cardiac Arrest & Resuscitation
438	Holinger	Pediatric Laryngology
443	Howard	Office Procedures in ENT
448	Juers	Hearing Tests
449	Keaster	Speech & Hearing in Children Under Five
454	Kos	Clinical Orientation in Management of Dizziness
459	Long	Chemotherapeutic & Antibiotic Agents
465	Moore	Diseases of Maxillary Sinus
475	Reger	Auditory Malingering
478	Roberts, G. J.	Management of Nasal Fractures
486	Shuster	Ocular Conditions of Otologic & Nasal Origin
491	Tremble	Nose & Throat Medication
492	Tschiassny	Locating Lesion in Facial Nerve Paralysis
497	Wilkerson	Hearing & Speech Center for Your City
498	Panel	Head Pain
501	Panel	Otological Problems
502	Panel	Medications

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Thursday

Key Th-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
406	Boyden	Chronic Frontal Sinus Surgery
408	Carhart	Audiogram in Otologic Diagnosis
410	Cohen	Rhinoplasty Complications
421	Fabricant	Modern Medication of Nose & Sinuses
423	Farrior & Carron	Fenestration Operation
432	Harrill	Bronchography
440	Holmes	Scars of Face
442	House	Surgery of Chronically Discharging Ear
445	Hunnicutt	Control of Hemorrhage
455	Kuhn	Allergic Diseases of Ear
456	LeJeune	Technic of Intralaryngeal Surgery
461	McConnell	Speech Audiometry for Otologist
463	McNally	Labyrinthine Tests
471	Perlman	Serous Otitis
476	Reger	Practical Office Audiometry
479	Roberts, S. E.	Head & Neck Syndrome
480	Ryan	Headache
481	Sataloff	Treatment of Nerve Deafness
483	Senturia	External Otitis
485	Shea	Intranasal Surgery
486	Shuster	Ocular Conditions of Otologic & Nasal Origin
488	Simonton	Recruitment of Loudness & Diplacusis Binauralis
492	Tschiassny	Locating Lesion in Facial Nerve Paralysis
495	Von Leden	Tracheotomy
503	Panel	Rhinology

Key Th-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
402	Ashley	Dust Therapy in Respiratory Allergy
413	Cottle	Septal Reconstruction Surgery
416	Cullen	Anesthesia
420	Erich	Fractures of Facial Bones
434	Hilger	Facial Fractures
436	Hill	Aural Rehabilitation
446	Jones	Tear Sac Surgery
447	Jordan	Otologic Conditions Complicated by Allergy
453	Kirchner	Difficulty in Swallowing
457	Lenz	Cancer of Nose & Throat: Radiotherapy
464	Miller	Laryngeal Emergencies in Infants & Children
467	Neivert	Care of Instruments
472	Pohlman	Plastic Ear Drum
489	Singleton	Tonsil & Adenoid Problem in Children
496	Walsh	Otitis Media: Treatment
499	Panel	Diseases of Larynx, Neck, Tracheobronchial Tree & Esophagus

Key Th-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
412	Costen	Mandibular Joint Syndrome
418	Derlacki	Allergy of Ear
419	DeWeese	The Dizzy Patient
428	Glorig	Evaluation of Methods for Measuring Auditory Acuity
430	Goodhill	Management of Tinnitus
439	Holinger	Esophageal Diseases in Infants & Children
450	Kelemen	Mechanical Injuries of Hearing Organ
452	Kinney	Acoustic Trauma
454	Kos	Clinical Orientation in Management of Dizziness
458	Lewin	Facial & Nasal Reconstructive Surgery
465	Moore	Diseases of Maxillary Sinus
469	Pastore	A Lump in the Throat
473	Pressman	Rhinologist & Problem of Rhinoplasty
474	Putney	Surgery of Neck Tumors
478	Roberts, G. J.	Management of Nasal Fractures
482	Seltzer	Plastic Surgery of Nose & Face-Lift Operation
484	Shambaugh	Failures in Fenestration Surgery
494	Van Alyea	Chronic Sinusitis
498	Panel	Head Pain
500	Panel	Tumors & Infections of Neck
502	Panel	Medications

News - - - Notes

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

The International Council of Ophthalmology has selected **Glaucoma** and **Uveitis** as the main subjects for discussion at the XVII International Congress of Ophthalmology, which will take place in New York, September 12-17, 1954. The names of the openers, also chosen by the International Council, will be announced soon.

Offers of papers and scientific exhibits are now being received at the office of the Secretary General of the Congress, Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minn.

PAN-AMERICAN ASSOCIATION OF OPHTHALMOLOGY

The Pan-American Association of Ophthalmology has announced that the officers of the V Congress of Ophthalmology, to be held in Santiago, Chile, in February 1956, will be: president, Dr. Cristobal Espildora; vice-president, Dr. Santiago Barrenechea; secretary general, Dr. Rene Contardo; and treasurer, Dr. Abraham Schweitzer.

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology has announced the following examinations: October 6 to 10, 1952, at the Palmer House in Chicago; April 21 to 25, 1953, at the Roosevelt Hotel in New Orleans.

AMERICAN COLLEGE OF SURGEONS

The Ophthalmology Programs for the 1952 New York Clinical Congress of the American College of Surgeons will include: 1. On Tuesday morning, September, 23, a panel discussion on "Problems Concerned with the Extraction of Complicated Cataracts." Moderator: John H. Dunnington, M.D., F.A.C.S., New York. Collaborators: Walter S. Atkinson, M.D., F.A.C.S., Watertown, N. Y.; Henry L. Birge, M.D., F.A.C.S., Hartford, Conn.; and Glen Gregory Gibson, M.D., Philadelphia.

2. On Tuesday evening, September 23, a symposium on "The Surgical Correction of Motor Anomalies Involving the Extraocular Muscles." The participants will be Harold W. Brown, M.D., New York; Harold G. Scheie, M.D., F.A.C.S., Philadelphia; Walter H. Fink, M.D., F.A.C.S., Minneapolis; and Raynold N. Berke, M.D., Hackensack, N. J.

3. On Wednesday morning, September 24, a panel discussion on "Orbital Implants." Moderator: Arno E. Town, M.D., F.A.C.S., New York. Collaborators: Alston Callahan, M.D., F.A.C.S., Birmingham, Ala.; John P. Macnie, M.D., F.A.C.S., New York; and Raymond E. Meek, M.D., F.A.C.S., New York.

4. On Wednesday evening, September 24, a symposium on "Causes of Failure in Ocular Surgical Procedures," with the following panel: Brittain Ford Payne, M.D., F.A.C.S., New York; Joseph A. C. Wadsworth, M.D., New York; Daniel B. Kirby, M.D., F.A.C.S., New York; Truman L. Boyes, M.D., F.A.C.S., New York; and P. Robb McDonald, M.D., F.A.C.S., Philadelphia.

5. On Thursday morning, September 25, a panel discussion on "Fallacies in Refraction By Use of Mechanical Refractors." Moderator: Conrad Berens, M.D., F.A.C.S., New York. Collaborators: Alfred Cowan, M.D., Philadelphia; Arthur Links, M.D., F.A.C.S., New York; and Daniel Syndacker, M.D., Chicago.

6. On Thursday evening, September 25, the following papers will be presented: "Sympathetic Ophthalmia," by Harold H. Joy, M.D., F.A.C.S., Syracuse, N. Y.; "Atomic Eye Injuries," by Elbert DeCoursey, Brigadier General, M.C., U. S. A., Washington, D. C.; "Managment of Intraocular Foreign Bodies," Raymond L. Pfeiffer, M.D., F.A.C.S., New York; and "Treatment of Eye Burns," By Robison D. Harley, M.D., F.A.C.S., Atlantic City, N. J.

AMERICAN OPHTHALMOLOGICAL SOCIETY

At the annual meeting of the American Ophthalmological Society the following officers were elected: president, Dr. Conrad Berens; vice-president, Dr. William L. Benedict; secretary-treasurer, Dr. Maynard C. Wheeler; and editor, Dr. Gordon M. Bruce.

AMERICAN OTOLOGICAL SOCIETY, INC.

At the annual meeting of the American Otological Society, Inc., on May 19, 1952, Dr. Albert C. Furstenberg was elected president for the year 1952-1953.

The next annual meeting of the American Otological Society, Inc., will be held in New Orleans, La., May 1 and 2, 1953, at the Roosevelt Hotel.

NATIONAL NOISE ABATEMENT SYMPOSIUM

The third annual National Noise Abatement Symposium will be held October 10 at Armour Research Foundation of Illinois Institute of Technology, Chicago.

The symposium will feature seven talks on various aspects of the noise problem by experts on the control, measurement, and effects of noise. Sponsors of last year's meeting were the Acoustical Society of America, The National Research Council, The National Noise Abatement Council, and the Foundation.

Daniel B. Callaway, supervisor of sound and vibration control at the Foundation, is chairman of the program committee for the meeting. Among the speakers at the afternoon session will be Dr. Howard P. House, University of Southern California, Los Angeles, who will speak on "The Ear and Noise"; Dr. Walter A. Rosenblith, Acoustics Laboratory, Massachusetts Institute of Technology, Cambridge, Mass., whose paper will deal with "The Effects of Noise on the Behavior of People"; Dr. Stacy R. Guild, Johns Hopkins Hospital, Baltimore, who will discuss "Safety Standards for Industrial Noise"; and Dr. Aram Glorig, Walter Reed Army Hospital, Washington, D.C., discussing "The Measurement of Hearing Loss."

OPTICAL SOCIETY OF AMERICA

The Thirty-Seventh Annual Meeting of the Optical Society of America will be held at the Hotel Statler in Boston, Mass., October 9-11, 1952. A special feature of this forthcoming meeting will be an address by the 1952 Ives Medalist, Dr. Ira S. Bowen of Mount Wilson and Palomar Observatories.

SOCIETY OF MILITARY OPHTHALMOLOGISTS

The Society of Military Ophthalmologists will hold its annual dinner meeting at the Palmer House, Chicago, Ill., at 6:30 p.m. on Tuesday, October 14, 1952, during the week of the meeting of the American Academy of Ophthalmology and Otolaryngology. Reservations should be made with the secretary, Colonel J. H. King, Jr., Walter Reed Army Hospital, Washington 12, D. C. Membership is limited to ophthalmologists on extended active duty with the Army, Navy and Air Forces.

ILLINOIS

The Central Illinois Society of Ophthalmology and Otolaryngology will meet at the Abraham Lincoln Hotel in Springfield, Ill., on September 12, 13 and 14. The guest speakers will be Dr. Paul A. Chandler of Boston, Mass. and Dr. Thomas C. Galloway of Evanston, Ill.

NEW YORK

The Medal for Outstanding Contribution to Ophthalmology in 1952, awarded by the American Medical Association, was presented to Dr. Arthur J. Bedell, Albany, at the recent meeting in Chicago, June 9 to 13.

PENNSYLVANIA

Dr. William E. Krewson, III, has recently been appointed Director of the Departments of Ocular Motility and Perimetry at the Wills Eye Hospital, Philadelphia.

Dr. Albert P. Seltzer of Philadelphia was awarded an honorary degree of Doctor of Laws by Shaw University, Raleigh, N.C., on May 26, 1952.

POSITIONS AVAILABLE

Research Fellowships in Ophthalmology: Information regarding research fellowships in ophthalmology at Indiana University Medical Center may be obtained by writing to Dr. T. F. Schlaegel, Jr., 1041 W. Michigan St., Indianapolis 7, Ind. It is not necessary for a candidate to have had training in ophthalmology to be accepted for one of these positions. For those who have not had a graduate course in ophthalmology, these research fellowships will provide some training prior to a residency. The fellows assist in the eye clinic and at eye surgery and attend the formal classes for residents in ophthalmology. The stipend is $150 a month plus laundry.

Positions available as associate surgeon, Division of Ophthalmology, Henry Ford Hospital. Personal interview required. Write E. L. Whitney, M. D., Henry Ford Hospital, Detroit, giving full particulars.

Residency in Ophthalmology at the George F. Geisinger Memorial Hospital and Foss Clinic, Danville, Pa. Applicant must have had previous ophthalmic training and/or postgraduate course in ophthalmology. Statistics for 1951: Out patients, 11,192; refractions, 5,480; consultations, 394; ophthalmic operations, 362; glasses released, 3,551; cataract extractions, 78; ophthalmic admissions, 285. Applications should be addressed to Clyde H. Jacobs, M.D., Director of the Department of Ophthalmology, Foss Clinic, Danville, Pa.

Listings of positions available should be sent to W. L. Benedict, M.D., 100 First Avenue Building, Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist; diplomate, resident, etc.); (2) type of position to be filled; and (3) whom to write for further information. Unless otherwise requested, listings will be published once only.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse or guarantee any of the published listings.

DIRECTORY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC SOCIETIES

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 47 Rue de Bellechasse, Paris, France
Secretary-General: Prof. A. Franceschetti, rue A—Jentzer 22, Geneva, Switzerland

International Council of Ophthalmology

President: Prof. F. W. Nordenson
Secretary: Prof. Dr. Edward Hartmann, 2, Avenue Ingres, Paris (16c) France

International Organization Against Trachoma

President: Dr. A. F. MacCallan
Secretary: Dr. F. Wibaut, P. C. Hoofstraat 145, Amsterdam, Holland

Pan-American Association of Ophthalmology

President: Dr. M. E. Alvaro
Secretary: Dr. Thomas D. Allen, 122 South Michigan Avenue, Chicago 2, Ill.
Time and Place: February 1956. Santiago, Chile

Pan-American Association of Ophthalmology, Puerto Rico Chapter

President: Dr. Luis J. Fernandez, Box 2206, San Juan 10, Puerto Rico
Secretary: Dr. P. Fernandez
Place: San Juan

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Jose C. Gros
Secretary: Dr. Pedro Hernandez Gonzalo, Calle 8, 358, Havana, Cuba

FOREIGN

The Ophthalmological and Otolaryngological Society of Alberta

President: Dr. W. E. Spankie
Secretary: Dr. M. R. Marshall, Wells Pavilion, University Hospital, Edmonton, Alberta, Canada
Time and Place: Spring of 1953 in Edmonton

All-India Ophthalmological Society

President: Dr. G. Zachariah
Secretaries: Dr. S. N. Cooper and Dr. V. K. Chitnis, Laud Mansion, Queen's Road, Bombay 4, India

Associacao Medica do Instituto Penido Burnier

President: Dr. Leoncio de Souza ueiroz
First Secretary: Dr. Milton B. de Toledo
Second Secretary: Dr. Aloisio Af. Ferreira

Austrian Otolaryngological Society, Vienna

President: Prof. Dr. E. Schlander
Secretary: Doz. Dr. O. Novotny, Vienna IX, Alserstrasse 4, Austria
Time: Annually

Belgian Ophthalmologic Society

President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, Avenue Ruelens 179, Louvain
Time and Place: January, June and November. Bruxelles

Bombay Ophthalmologists' Association

Chairman: Rotated
Conveners: Dr. S. N. Cooper and Dr. B. D. Telang, Laud Mansion 21, Queen's Road, Bombay 4
Time and Place: 7:30 p.m., third Wednesday of each month. The seven ophthalmic hospitals of Bombay by rotation

British Association of Otolaryngologists

President: Mr. V. E. Negus
Honorary Secretary: Mr. F. C. W. Capps, 45 Lincoln's Inn Fields, London W. C. 2, England

British Medical Association, Section on Ophthalmology

President: Mr. O. G. Morgan
Secretary: Mr. A. G. Cross, 27 Harley Street, London W. 1, England

Centro de Estudos de Oftalmologia de Sao Paulo

President: Dr. Rubens Belfort Mattos
Secretary: Dr. Jose Carlos Gouvea Pacheco, Marconi 34, Sao Paulo

Chengtu Ophthalmological Society

President: Dr. Eugene Chan
Secretary: Dr. D. S. Shen, Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China

Chinese Ophthalmology Society

President: Dr. C. H. Chou
Secretary: Dr. F. S. Tsang, 221 Foochow Road, Shanghai

Chinese Ophthalmological Society of Peking

President: Dr. G. C. Lin
Secretary: Dr. H. L. Chen, Ophthalmological Dept., Peking University Medical School, Peking
Time: Bimonthly

Deutsche Ophthalmologische Gesellschaft Heidelberg

President: Prof. Dr. med. K. Wessely
Secretary: Prof. Dr. med. E. Engelking, Heidelberg, Universitäts-Augenklinik

Faculty of Ophthalmologists

President: Mr. O. M. Duthie
Secretary: Mr. J. H. Doggart, F.R.C.S., 45 Lincoln's Inn Fields, London, W.C. 2, England

French Society of Ophthalmology

Secretary: Dr. Edward Hartmann, 2 Ave Ingres, Paris 16, France

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Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

GERMAN OPHTHALMOLOGICAL SOCIETY

President: Prof. W. Lohlein
Secretary: Prof. E. Engelking, Heidelberg

HUNGARIAN MEDICAL TRADE UNION, SECTION OF OPHTHALMOLOGY

President: Prof. I. Csapody
Secretary: Dr. E. Galla, Krisztina Körut 139, Budapest, Hungary
Time and Place: Bimonthly. Second Eye Clinic, Budapest

HUNGARIAN OPHTHALMOLOGICAL SOCIETY

President: Prof. G. Horay
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, No. 1, Illesucca 15, Budapest

ISRAEL OPHTHALMOLOGICAL SOCIETY

President: Dr. Aryeh Feigenbaum
Secretary: Dr. E. Sinai, 9 Bialik Street, Tel Aviv

MIDLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. F. A. Anderson
Secretaries: Dr. P. Jameson Evans, 51 Calthorpe Road, Edgbaston, Birmingham 15, England
Dr. R. D. Weeden Butler, 18 Highfield Road, Edgbaston, Birmingham 15, England
Place: Birmingham and Midland Eye Hospital, Church Street, Birmingham 3, England

NEDERLANDSCH OOGHEELKUNDIG GEZELSCHAP

President: Prof. A. W. Mulock Houwer
Secretary: Dr. T. A. Vos, Laan v. Meerdervoort 394, the Hague

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Mr. A. McKie Reid
Secretary: Mr. W. M. Muirhead, 70 Upper Hanover Street, Sheffield 3, England
Time and Place: October to May. Manchester, Leeds, Newcastle-on-Tyne, Liverpool, Sheffield and Bradford

SOCIETY OF NORWEGIAN OPHTHALMOLOGISTS

President: Dr. J. C. Holst, Pilestredet 28, Oslo
Secretary: Dr. Thore Lie Thomassen
Place: Rikshopitalets Oeyenklinikk, Oslo

THE NOVA SCOTIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. K. Fuller
Secretary-Treasurer: Dr. E. I. Glenister, 513 Barrington Street, Halifax, Nova Scotia
Time: Four times yearly at dates to be arranged by the Executive

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA

President: Dr. G. A. Brew
Secretary: Dr. Arnold L. Lance, 135 Macquarie Street, Sydney, New South Wales
Time and Place: August 1953, in Melbourne

OPHTHALMOLOGICAL SOCIETY OF COPENHAGEN

President: Prof. Dr. Holger Ehlers
Secretary: Dr. Carl Johan Moellenbach, Rigshospitalets oejenafdeling, Copenhagen O, Denmark
Place: Rigshospitalets oejenafdeling

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Dr. Ibrahim Ahmad Mohammad
Secretary: Dr. Mahmoud Lutfi, Ophthalmic Hospital, Giza
Time and Place: Annual meeting during March. Summer meeting during August. Dar El Hekmah, 42 Kasr El Ainy Street, Cairo, Egypt

OPHTHALMOLOGICAL SOCIETY OF HOSPITAL DE NUESTRA SENORA DE LA LUZ

Chairman: Dr. Manuel J. Icaza y Dublan
Secretary: Dr. Jorge Meyran, Ezequiel Montes 135, México, D. F.
Time and Place: Second Friday of each month. Hospital de Nuestra Senora de la Luz

OPHTHALMOLOGICAL SOCIETY OF NEW ZEALAND

President: Dr. L. S. Talbot
Secretary: Dr. W. J. Hope-Robertson, Kelvin Chambers, 16 The Terrace, Wellington
Time and Place: Annually. Auckland, Wellington, Christchurch and Dunedin alternately

OPHTHALMOLOGICAL SOCIETY OF SOUTH AFRICA

President: Dr. L. Staz
Secretary-Treasurer: Dr. J. Guillaume Louw, 901 Dumbarton House, Adderley Street, Cape Town
Time: Annually

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Dr. A. MacRae
Honorary Secretaries: Mr. A. G. Leigh and Mr. Harold Ridley, 45 Lincoln's Inn Fields, London

OPHTHALMOLOGISCHE GESELLSCHAFT IN WIEN

President: K. Lindner
Secretary: Ch. Eyb, Wien, IX, Alserstrasse 4

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. F. A. Anderson
Hon. Secretary and Treasurer: Ian C. Fraser, F.R.C.S., Red Roofs, Kingsland, Shrewsbury

PHILIPPINE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Jesus Eusebio
Secretary-Treasurer: Dr. Jesus Tamesis, Philippine General Hospital, Manila

POLISH OPHTHALMOLOGICAL SOCIETY

President: Prof. Dr. W. Kapuscinski
Secretary: Dr. S. Topolski, Piusa 38, Warsaw, Poland
Time and Place: Every two years—summer. Ophthalmic Clinic, Oczki 6, Warsaw

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Montague Hine, F.R.C.S.
Secretaries: Arthur Lister, F.R.C.S., 56 Wimpole Street, London, W.1., England
H. E. Hobbs, F.R.C.S., 129 Harley Street, London, W.1., England

SAO PAULO SOCIETY OF OPHTHALMOLOGY
President: Dr. Durval Prado
Secretary: Dr. Rubens Belfort Mattos, Rua B. Stapetininga, 29 F-3° andar, Sao Paulo, Brazil

SCOTTISH OPHTHALMOLOGICAL CLUB
President: Dr. E. H. Cameron
Secretary: Dr. John Marshall, 11 Clairmont Gardens, Glasgow, C.3, Scotland
Time and Place: Last Saturday of March and October, Edinburgh and Glasgow, in rotation

SOCIEDAD ARGENTINA DE OFTALMOLOGIA
President: Dr. Jorge Malbran
Secretary: Dr. Jose Maria Roveda, Lezica 4472, Buenos Aires
Time and Place: Third Wednesday of every month. Buenos Aires

SOCIEDAD CHILENA DE OFTALMOLOGIA
President: Dr. Italo Martini
Secretary: Dr. Adrian Araya Costa, Renaca, no. 34, Santiago, Chile

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y DE OTORHINOLARINGOLOGIA
President: Dr. Jorge Suarez-Hoyos, Carrera 5a, no. 13-39, Bogota, Colombia
Secretary: Dr. Francisco Arango
Time and Place: Second Tuesday of each month. Club Medico

SOCIEDAD CUBANA DE OFTALMOLOGIA
President: Dr. Oscar F. Horstmann
Secretary: Dr. Heriberto Buch Granados, Calle B No. 668 ent. 27 y 29, Vedado, Habana, Cuba
Time and Place: First Thursday bi-monthly. Malecon No. 61 Bajos, Havana, Cuba

SOCIEDAD MEXICANA DE OFTALMOLOGIA
President: Dr. Teodulo M. Agundis
Secretary: Dr. Jose Luis Arce, Viena 3-5, Mexico D. F., Mexico
Time and Place: 8:30 p.m., first Tuesday of each month, Gral. PRIM 47

SOCIEDAD NICARAGUENSE DE OFTALMOLOGIA
President: Dr. Ricardo Lacayo
Secretary: Dr. Gilberto Cuadra, Clinica Especializada, Managua, D. N., Nicaragua, C. A.

SOCIEDAD DE OFTALMOLOGIA DE CORDOBA
Chairman: Dante J. Yadarola
Secretary: Dr. Alberto Urrets Zavalia (hijo), 27 de Abril 255, Cordoba, Argentina

SOCIEDAD DE OFTALMOLOGIA DE GUADALAJARA
President: Dr. Elias Mendoza Gonzalez
Secretary: Dr. Jose Martin del Campo, Av. Juarez 211, Desp. 314, Guadalajara, Jal.
Place: Edificio Lutencia Despacho 101

SOCIEDAD OFTALMOLOGICA HISPANO AMERICANA
President: Dr. Soria
Secretary: Dr. Costi, 3. Calle Montalban, Madrid

SOCIEDAD DE OFTALMOLOGIA DEL LITORAL
President: Dr. Juan Manuel Vila Ortiz, Cordoba 1915, Rosario, Argentina
Secretary: Dr. Carlos M. Soto
Time and Place: Last Sunday in every month. Rosario

SOCIEDAD OFTALMOLOGICA DE VALPARAISO
President: Prof. Dr. Jean H. Thierry
Secretary-Treasurer: Dr. Jorge Oyarzun O, Hospital de Ojos, Calle Blas, Cuevas, Valparaiso

SOCIEDAD PERUANA DE OTO-RINO-LARINGOLOGIA Y OFTALMOLOGIA
President: Dr. Enrique Cipriani V., Pasaje Olaya 156, Lima, Peru
Secretario General: Dr. Enrique Haro
Secretario de Actas: Dr. Alberto Castillo R.

SOCIEDADE BRASILEIRA DE OFTALMOLOGIA
President: Dr. Paulo C. Pimental
First Secretary: Dr. Pedro Moacyr de Aguiar, Ladeira Santa Thereza 136, Apt. 101, Rio de Janeiro
Second Secretary: Dr. Luiz Enrico Ferreira
Time: Third Friday of every month from April to December

SOCIEDADE DE OFTALMOLOGIA DEL NORTE
President: Dr. Jorge Luis Castillo
Secretary: Dr. Felix Berman, San Lorenzo 345, Tucuman, Argentina
Place: Mendoza 421, Tucuman

SOCIEDADE DE OFTALMOLOGIA DE MINAS GERAES
President: Dr. Amelio Bonfioli
Secretary: Dr. Mariana Noronha, Pauso Alegre 1150, Belo Horizonte, Est. Minas Geraes

SOCIEDADE DE OFTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DE BAHIA
President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil
Secretary: Dr. Adroaldo de Alencar

SOCIEDADE DE OFTALMOLOGIA E OTO-RINO-LARINGOLOGIA DO RIO GRANDE DO SUL
President: Dr. Mario Araujo Azambuja
First Secretary: Dr. Humberto Lubisco, Rua Donna Laura 45, Porto Alegre, Est., Rio Grande do Sul
Second Secretary: Dr. Ivo Kuhl

SOCIEDADE DE OFTALMOLOGIA DE SAO PAULO
President: Dr. Paulo Braga Magalhaes
General Secretary: Dr. Paulo Aranha de Azevedo, Xavier de Toledo 71,1° and., Sao Paulo
First Secretary: Dr. Alfredo Rocco

SOCIEDADE DE OTO-RINO-LARINGOLOGIA DO RIO DE JANIERO
President: Dr. Aloysio Novis
Secretaries: Dr. Rubens Cabral, Rua Paissandu 73, Rio de Janeiro, Brazil
Dr. Mairelles Vieira

SOCIETA OFTALMOLOGICA ITALIANA

President: Prof. Giuseppe Ovio
Secretary: Prof. E. Leonardi, Piazza degli Eroi, 11 Roma, Italy
Place: Roma Piazza degli Eroi 11

SOCIETE BELGE D'OPHTALMOLOGIE

President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, 179 avenue Reulens, Louvain, Belgium
Time: Last Sunday of February, June and November

SOCIETE D'OTO-RHINO-LARYNGOLOGIE

Administrateur: G. Raimond, 17et19, Rue de Buci, Paris (VIe) France

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. S. Larsson
Secretary: Dr. K. O. Granstrom, Sodermalmstorg 4, Stockholm, Sweden

SOUTHERN OPHTHALMOLOGICAL SOCIETY, ENGLAND

President: Campbell Shaw, M. R. C. S.
Hon. Secretary and Treasurer: Nigel Cridland, D.M., D.O., 25 Craneswater Park, Southsea, England

NATIONAL

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. James Milton Robb
Executive Secretary-Treasurer: Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minn.
Time and Place: October 12-17, 1952, Chicago

AMERICAN ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES

President: Dr. M. E. Alvaro
Secretary-Treasurer: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. Gordon B. New
Secretary: Dr. Louis H. Clerf, 1530 Locust, Philadelphia 2, Pa.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. C. Stewart Nash
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester 7, N. Y.

EASTERN SECTION
Vice-President: Dr. Francis W. Davison
Secretary: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.

MIDDLE SECTION
Vice-President: Dr. Ralph J. McQuiston
Secretary: Dr. Walter H. Theobald, 307 North Michigan Avenue, Chicago 1, Ill.

SOUTHERN SECTION:
Vice-President: Dr. Lester A. Brown
Secretary: Dr. James W. McLaurin, Raymond Building, Baton Rouge 6, La.

WESTERN SECTION:
Vice-President: Dr. Howard P. House
Secretary: Dr. Harold Boyd, 1136 West Sixth Street, Los Angeles, Calif.

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Edwin B. Dunphy
Secretary: Dr. Trygve Gundersen, 101 Bay State Road, Boston, Mass.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. James Milton Robb
Secretary: Dr. Sam H. Sanders, 1089 Madison Avenue, Memphis 3, Tenn.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Conrad Berens
Secretary-Treasurer: Dr. Maynard C. Wheeler, 30 W. 59th Street, New York 19, N. Y.
Time and Place: May 28-30, 1953, The Homestead, Hot Springs, Va.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Albert C. Furstenberg
Secretary: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill.
Time and Place: May 1-2, 1953, Roosevelt Hotel, New Orleans, La.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY

President: Dr. Norman N. Smith
Secretary: Dr. Joseph G. Gilbert, 1404 President Street, Brooklyn 13, N. Y.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. William H. Evans
Secretary-Treasurer: Dr. Joseph W. Hampsey, 806 May Building, Pittsburgh 22, Pa.
Time and Place: Annual meeting, October 17, 1952, Chicago

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. Walter H. Fink
Secretary-Treasurer: Dr. James H. Allen, 1430 Tulane Avenue, New Orleans, La.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. R. G. C. Kelly
Secretary: Dr. J. Clement McCulloch, 380 Medical Arts Building, Toronto, Ont.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. R. F. Nicholls
Secretary: Dr. J. F. A. Johnston, 174 St. George Street, Toronto, Ont.

CANADIAN OTOLARYNGOLOGICAL SOCIETY

President: Dr. Jules Brahy
Secretary: Dr. W. Ross Wright, 361 Regent Street, Fredericton, New Brunswick

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS, INC.
President: Mr. Mason H. Bigelow
Secretary: Dr. Franklin M. Foote, 1790 Broadway, New York 19, N. Y.

REGIONAL

ARK-LA-TEX OTO-OPHTHALMIC SOCIETY
President: Dr. Joel A. Wilkinson
Secretary: Dr. Frank L. Bryant, 2622 Greenwood, Shreveport, La.
Time and Place: First Monday of month, September through June. Shreveport Club

HAWAII EYE, EAR, NOSE AND THROAT SOCIETY
Chairman: Dr. John P. Frazer
Secretary: Dr. C. W. Trexler, Young Hotel Building, Honolulu, T. H.
Time and Place: Each Thursday (dinner meetings) 6:30 p.m., Pacific Club, Honolulu

INTER-MOUNTAIN OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. Harry O. Frazier
Secretary: Dr. Robert L. Rees, 115 East South Temple Street, Salt Lake City, Utah
Time and Place: Third Monday of each month, September through May. University Club, 136 East South Temple, Salt Lake City

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President Dr. W. L. Hughes
Secretary: Dr. Edley H. Jones, 1301 Washington Street, Vicksburg, Miss.
Time and Place: May 11, 1953, Edgewater Gulf Hotel, Edgewater, Miss.

NASSAU OPHTHALMOLOGICAL SOCIETY
President: Dr. A. Milton Goldman
Secretary: Dr. Ward L. Mould, 131 Fulton Avenue, Hempstead, N. Y.
Time and Place: Fourth Monday in February, April, September and November at 7:30 p.m. at Meadowbrook Hospital, Hempstead, N. Y.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY
President: Dr. Benjamin A. Sachs
Secretary: Dr. Garrett L. Sullivan, 101 Bay State Road, Boston 15, Mass.
Time and Place: Third Wednesday of each month, November through April. Massachusetts Eye and Ear Infirmary

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. Leighton F. Johnson
Secretary-Treasurer: Dr. Burton E. Lovesey, 76 Bay State Road, Boston 15, Mass.
Time and Place: Quarterly. Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. C. Allen Dickey
Secretary-Treasurer: Dr. Howard P. House, 1136 West Sixth Street, Los Angeles 14, Calif.
Time and Place: 1953, Los Angeles, Calif.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. William B. Hubbard
Secretary: Dr. Frank A. Ware, 514 Genesee Bank Bldg., Flint, Mich.
Time and Place: Second Tuesday evening of each month, except summer months. Bancroft Hotel, Saginaw

SIOUX VALLEY EYE AND EAR ACADEMY
President: Dr. James Reeder, Jr.
Secretary-Treasurer: Dr. W. P. Davey, 627-632 Frances Building, Sioux City, Iowa

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Edley H. Jones
Secretary. Dr. F. A. Holden, Medical Arts Building, Baltimore, Md.
Time and Place. November 10-13, 1952, Miami, Fla.

TIDEWATER OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. C. C. Cooley
Secretary. Dr. J. G. Sellers, 413 Medical Arts Building, Norfolk, Va.

WISCONSIN-UPPER MICHIGAN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. H. D. McEachran
Secretary: Dr. G. L. McCormick, 650 South Central Avenue, Marshfield, Wis.
Time and Place: September 6-7, 1952, Maxwelton Braes, Sturgeon Bay, Wis.

STATE

ALABAMA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Ralph Clements
Secretary-Treasurer: Dr. David A. McCoy, 501 Woodward Building, Birmingham, Ala.

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION
President: Dr. C. G. Hinkle
Secretary: Dr. K. W. Cosgrove, 113 East Capitol Avenue, Little Rock, Ark.

CALIFORNIA MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION
Chairman: Dr. Robert C. McNaught
Secretary: Dr. Alfred R. Robbins, 1930 Wilshire Blvd., Los Angeles, Calif.

COLORADO OPHTHALMOLOGICAL SOCIETY
President: Dr. John Bradley
Secretary: Dr. Robert C. Shattuck, 406 Republic Building, Denver, Colo.
Time and Place: First Saturday of each month, October through May, Colorado School of Medicine, Denver

COLORADO OTOLARYNGOLOGICAL SOCIETY
President: Dr. Ivan E. Hix
Secretary: Dr. Merrill O. Dart, 3191 S. Broadway, Englewood, Colo.
Time and Place: First Saturday of each month, October through May. Colorado University School of Medicine

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Charles C. Grace
Secretary-Treasurer: Dr. Carl S. McLemore, 1202 South Kuhl Avenue, Orlando, Fla.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Stacy C. Howell
Secretary: Dr. Alton V. Hallum, 245 Doctors Building, Augusta 3, Ga.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. E. L. Van Buskirk
Secretary-Treasurer: Dr. John R. Swan, 23 East Ohio Street, Indianapolis 4, Ind.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Byron Merkel
Secretary: Dr. Carl A. Noe, 601-611 Highley Building, Cedar Rapids, Iowa

KANSAS STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. N. L. Francis
Secretary: Dr. W. D. Pitman, First National Bank Building, Pratt, Kan.

MASSACHUSETTS MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Edwin B. Dunphy
Secretary: Dr. John E. McKeigue, 403 Commonwealth Avenue, Boston 15, Mass.

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. J. E. Croushore (Otolaryngology)
Co-Chairman: Dr. J. C. Gemeroy (Ophthalmology)
Secretary: Dr. R. W. Teed, 215 South Main, Ann Arbor, Mich. (Otolaryngology)
Co-Secretary: Dr. F. B. Heckert, 1105 Bank of Lansing Building, Lansing, Mich. (Ophthalmology)

MICHIGAN TRIOLOGICAL SOCIETY
President: Dr. William D. Irwin
Secretary: Dr. V. E. Cortopassi, 324 South Washington Avenue, Saginaw, Mich.
Time: Second Thursday of November, December, March, April and May

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. K. A. Phelps
Secretary-Treasurer: Dr. R. W Holmen, Lowry Medical Arts Building, St. Paul 2, Minn.
Time and Place: Second Friday of each month, November through May. Alternating, Minnesota Club, St. Paul, and Minneapolis Club, Minneapolis

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY
President: Dr. Fritz D. Hurd
Secretary-Treasurer: Dr. H. L. Casebeer, Pennsylvania Building, 44 West Park Street, Butte, Mont.
Time and Place: September 19, 1952, Missoula, Mont.

NEW JERSEY OPHTHALMOLOGICAL SOCIETY
President Dr. A. M. K. Maldeis
Secretary: Dr. A. Russell Sherman, 671 Broad Street, Newark, N. J.

NEW MEXICO OPHTHALMOLOGIC SOCIETY
President: Dr. A. L. Schonberg
Secretary: Dr. Howard Peck, Medical Arts Square, Albuquerque, N. M.
Time: Meetings four times a year

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. MacLean B. Leath
Secretary-Treasurer: Dr. George B. Ferguson, 1110 West Main St., Durham, N. C.
Time and Place: September 15-17, 1952, Hotel Robert E. Lee, Winston-Salem, N. C.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Richard S. Fixott
Secretary: Dr. George E. Chamberlain, 909 Medical Dental Building, Portland 5, Ore.
Time and Place: Third Tuesday each month September through May. Old Heathman Hotel, Portland

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Samuel T. Buckman
Secretary: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.
Time: Annually

PUERTO RICO MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Luis J. Fernandez
Secretaries: Dr. Ricardo F. Fernandez, P.O. Box 2206, San Juan 10, Puerto Rico (Ophthalmology)
Dr. Carlos E. Munoz MacCormick, P.O. Box 604, San Juan, Puerto Rico (Otolaryngology)
Time and Place: Bimonthly. Puerto Rico Medical Association Building

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Thomas R. Gaines
Secretary-Treasurer: Dr. Roderick Macdonald, 330 East Main Street, Rock Hill, S. C.
Time and Place: September 15-17, 1952, Hotel Robert E. Lee, Winston-Salem, N. C.

TENNESSEE STATE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Roland H. Myers
Secretary-Treasurer: Dr. John T. Bryan, 2105 Hayes Street, Nashville, Tenn.
Time: Annually

TEXAS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. J. Charles Dickson
Secretary: Dr. Lyle Hooker, Esperson Building, Houston, Texas

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. A. D. Morgan
Secretary-Treasurer: Dr. G. Slaughter Fitz-Hugh. 104 East Market St., Charlottesville, Va.
Time and Place: Fall Clinical Session: Ophthalmology, November 18-19, 1952; Otolaryngology, November 20-21, 1952, Charlottesville, Va.; Spring meeting, May 1953, Hot Springs, Va.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Melvin K. McGehee
Secretary-Treasurer: Dr. Frederick C. Reel, Medical Arts Building, Charleston, W. Va.
Time and Place: May 2-3, 1953, Hot Springs, Va. Joint meeting with Virginia Society

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. William F. Krone
Secretary: Dr. Francis J. Grant, 1224 Salem Avenue, Hillside, N. J.
Time and Place: 8:45 p.m., second Monday of the month. The Academy of Medicine, 91 Lincoln Park, South, Newark

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. C. R. Anderson
Secretary-Treasurer: Dr. A. L. Peter, 406 Akron Savings and Loan Building, Akron, Ohio
Time and Place: 6:30 p.m., first Monday in January. March, May and November. Akron City Club

ALUMNI ASSOCIATION NEW YORK EYE AND EAR INFIRMARY
President: Dr. W. T. Boland
Secretary-Treasurer: Dr. Hunter Romaine, 111 East 65th Street, New York, N. Y.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. F. Phinizy Calhoun, Jr.
Secretary-Treasurer: Dr. James T. King, 384 Peachtree Street N.E., Atlanta 3, Ga.
Time and Place: 7:30 p.m., fourth Monday of each month, October to May. Academy of Medicine

BALTIMORE CITY MEDICAL SOCIETY, OPHTHALMOLOGICAL SECTION
Chairman: Dr. M. Elliott Randolph
Secretary-Treasurer: Dr. Abraham Kremen, 2355 Eutaw Place, Baltimore 17, Md.
Time and Place: 8:00 p.m., fourth Thursday of every month October to May. Medical and Chirurgical Faculty, 1211 Cathedral St.

BIRMINGHAM EYE, EAR, NOSE AND THROAT SOCIETY
President: Each member, in alphabetical order.
Secretary: Dr. David A. McCoy, Woodward Building, Birmingham, Ala.
Time and Place: 6:30 p.m., second Tuesday of each month, September through May. Thomas Jefferson Hotel

BOSTON CITY HOSPITAL AURAL AND OPHTHALMIC ASSOCIATION
President: Dr. Joseph Nerbonne
Secretary: Dr. Benjamin Riseman, 41 Bay State Road, Boston 15, Mass.

BROOKLYN OPHTHALMOLOGICAL SOCIETY
President: Dr. Daniel K. Kravitz
Secretary-Treasurer: Dr. Louis Freimark, 256 Rochester Avenue, Brooklyn 13, N. Y.
Time and Place: 8 p.m., third Thursday in October, December, February and April. Medical Society of the County of Kings, 1313 Bedford Avenue

BUFFALO OPHTHALMOLOGIC CLUB
President: Dr. Milton A. Palmer
Secretary: Dr. Elizabeth P. Olmsted, 568 Lafayette Avenue, Buffalo 22, N. Y.
Time and Place: Second Thursday of each month, October through May. Park Lane

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. G. C. Otrich
Secretary-Treasurer: Dr. Alfred G. Schultz, 237 East State Street, Jacksonville, Ill.
Time and Place: September 12-14, 1953, Abraham Lincoln Hotel, Springfield, Ill.

CENTRAL NEW YORK EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Donald E. Moore
Secretary-Treasurer: Dr. Lee R. Stoner, 1100 East Genesee Street, Syracuse 10, N. Y.

CHATTANOOGA EYE, EAR, NOSE AND THROAT SOCIETY
Chairman and Secretary: Dr. Willard H. Steele, Jr., 552 McCallie Avenue, Chattanooga, Tenn.
Time and Place: 6:30 p.m., fourth Thursday in each month, October through May. Mountain City Club

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY
President: Dr. Arthur J. Coombs
Secretary: Dr. Lawrence J. Lawson, 636 Church Street, Evanston, Ill.

CHICAGO OPHTHALMOLOGICAL SOCIETY
President: Dr. William F. Hughes, Jr.
Secretary-Treasurer: Dr. Gail R. Soper, 636 Church Street, Evanston, Ill.
Time and Place: 7:30 p.m., third Monday of each month, October to May. Chicago-Illini Union, 715 S. Wood Street

CINCINNATI OPHTHALMOLOGIC CLUB
Presidium: Dr. Donald J. Lyle, Dr. Karl W. Ascher, Dr. Josef D. Weintraub
Secretary: Dr. Josef D. Weintraub, 715 Provident Bank Building, Seventh and Vine Streets, Cincinnati, Ohio
Time and Place: Second Wednesday night of month, November through May. University Club, Fourth and Broadway

CINCINNATI OTOLARYNGOLOGIC SOCIETY
President: Dr. Raymond L. Hilsinger
Secretary-Treasurer: Dr. Stephen P. Hogg, 556 Doctors' Building, Cincinnati 2, Ohio
Time and Place: 6:30 on the third Monday of each month except July, August, September and December. University Club.

CLEVELAND OPHTHALMOLOGICAL CLUB
President: Dr. Lorand V. Johnson
Secretary-Treasurer: Dr Irwin H. Stolzar, 831 National City Bank Building, Cleveland 14, Ohio
Time and Place: Second Tuesday of November, January, February and April. University Club, Cleveland, Ohio

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY
Chairman: Dr. George F. J. Kelly
Clerk: Dr. M. Luther Kauffman, Medical Arts Building, Jenkintown, Pa.
Time and Place: 8:15 p.m., third Thursday of every month, October through April. College of Physicians Building

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Rex E. House
Secretary-Treasurer: Dr. L. David Wright, Thomas-Spann Clinic, 1546 South Brownlee, Corpus Christi, Texas
Time and Place: First Wednesday of each month. La Louisiane

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. W. B. Wilkinson
Secretary: Dr. Claude D. Winborn, Medical Arts Building, Dallas 1, Texas
Time and Place: First Tuesday of each month. Melrose Hotel

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. J. H. McNamee
Secretary-Treasurer: Dr. H. H. Gurau, 213 Bankers Trust Building, Des Moines, Iowa
Time and Place: 6:00 p.m., fourth Monday of each month, September through May. Des Moines Club

DETROIT OPHTHALMOLOGICAL CLUB
Secretary-Treasurer: Dr. Frederick A. Lauppe, 1249 David Whitney Building, Detroit 26, Mich.

DETROIT OPHTHALMOLOGICAL SOCIETY
President: Dr. Arthur S. Hale
Secretary: Dr. Arthur P. Wilkinson, 974 Fisher Building, Detroit 2, Mich.
Time and Place: Fourth Thursday of each month, November through April, at L'Aiglon, Fisher Building

DETROIT OTO-LARYNGOLOGICAL SOCIETY
President: Dr. B. F. Glowacki
Secretary-Treasurer: Dr. John R. Birch, 1010 Maccabees Building, Detroit 2, Mich.
Time and Place: Third Wednesday of each month, September to May. Wayne County Medical Society Building

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION
President: Dr. Harry Miller
Secretary-Treasurer: Dr. Frank Furlong, 713 Union Street, Schenectady, N. Y.
Time and Place: 8 p.m., first Thursday of month, October to June. Albany, Troy, Schenectady, rotating monthly

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Hal Maxwell
Secretary: Dr. William Skokan, 1708 Medical Arts Building, Fort Worth 2, Texas
Time and Place: 6:30 p.m., first Friday of each month, except July and August. St. Joseph's Hospital

HOUSTON ACADEMY OF MEDICINE, OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SECTION
President: Dr. Lyle Hooker
Secretary: Dr. Claude C. Cody, III, 1304 Walker Avenue, Houston 2, Texas
Time: Second Thursday of each month, October through June

EYE, EAR, NOSE AND THROAT SOCIETY OF HUDSON COUNTY
President: Dr. A. V. Saradarian
Secretary-Treasurer: Dr. A. J. Magee, 921 Bergen Avenue, Jersey City 6, N. J.
Time and Place: Second Monday of each month (September through May), Jersey City Medical Center

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. Marlow W. Manion
Secretary-Treasurer: Dr. Lewis E. Morrison, 603 Hume Mansur Building, Indianapolis 4, Ind.
Time and Place: 6:30 p.m., second Thursday of each month, November to May. Indianapolis Athletic Club

KANSAS CITY SOCIETY OF O. O. R. L.
President: Dr. A. W. McAlester, III
Secretary: Dr. Barnard C. Trowbridge, 411 Alameda Road, Kansas City 2, Mo.
Time and Place: Third Thursday of the month, November through May. Hotel President, 14th and Baltimore

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Edmund D. Godwin
Secretary: Dr. James V. Keipp, 110 Pine Avenue, Long Beach 2, Calif.
Time and Place: 6:00 p.m., monthly, September through May. Seaside Memorial Hospital

LOS ANGELES OPHTHALMOLOGICAL SOCIETY
President: Dr. Deane C. Hartman
Secretary: Dr. Daniel B. Esterly, 104 North Madison Avenue, Pasadena 1, Calif.
Time and Place: First Thursday of each month, September through June Los Angeles County Medical Society Building, 1925 Wilshire Boulevard

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Victor Goodhill
Secretary: Dr. Orwyn Ellis, 523 West Sixth Street, Los Angeles 14, Calif.
Time and Place: 6:00 p.m., fourth Monday of each month, September through May. Los Angeles County Medical Association Building, 1925 Wilshire Boulevard

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION ON OPHTHALMOLOGY
President: Dr. Joseph Dessoff
Secretary-Treasurer: Dr. J. Spencer Dryden, 1835 Eye Street Northwest, Washington 6, D. C.
Time: November 4, 1952; January 6 and May 5, 1953

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION ON OTOLARYNGOLOGY
Chairman: Dr. Irvin Feldman
Secretary: Dr. John Louzan, 1801 Eye Street N. W., Washington 6, D. C.
Time and Place: 7:00 p.m., third Tuesday in October, November, January, March and May. Army and Navy Club, 17th and Farragut Square, N.W.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Each member, in alphabetical order
Secretary: Dr. Roland H. Myers, 1720 Exchange Building, Memphis, Tenn.
Time and Place: Second Tuesday in each month, September through May. Memphis Eye, Ear, Nose and Throat Hospital

MILWAUKEE OTO-OPHTHALMIC SOCIETY
President: Dr. J. P. Wild
Secretary-Treasurer: Dr. Howard High, 324 East Wisconsin Avenue, Milwaukee 2, Wis.
Time and Place: 6:30 p.m., fourth Tuesday of each month, October to May. Athletic Club

MONTGOMERY COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
President: Dr. Robert A. Bruce
Secretary-Treasurer: Dr. L. N. Shroder, 144 West Fourth Street, Greenville, Ohio
Time and Place: First Tuesday of November, February, March and May. Dayton Country Club

MONTREAL OPHTHALMOLOGICAL SOCIETY
President: Dr. Jules Brahy
Secretary-Treasurer: Dr. Leo S. S. Kirschberg, 1390 Sherbrooke Street West, Montreal, Que., Canada
Time: Second Thursday of October, December, February and April

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. John W. Harned, Jr.
Secretary-Treasurer: Dr. Herbert Duncan, Bennie Dillon Building, Nashville, Tenn.
Time and Place: Third Monday evening of month, October through May. James Robertson Hotel

NEW YORK ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY
Chairman: Dr. James W. Smith
Secretary: Dr. A. Gerard De Voe, 30 West 59th St., New York, N. Y.
Time and Place: Third Monday of the month, October through May. New York Academy of Medicine

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY
President: Dr. Abraham L. Kornzweig
Secretary: Dr. Leon H. Ehrlich, 211 Central Park West, New York 24, N. Y.
Time and Place: First Monday of the month, October to May. New York Academy of Medicine

NUECES COUNTY EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. C. N. Meador
Secretary: Dr. Rex C. House, 228 Medical-Dental Building, Corpus Christi, Texas
Time and Place: 7:30 p.m., second Monday of each month, October through May. 'Dusty's, 1739 South Brownlee Street, Corpus Christi, Texas

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. C. W. McClure
Secretary: Dr. Richard A. Clay, 416 Northwest 13th Street, Oklahoma City, Okla.
Time and Place: First Tuesday of each month. University Hospital, 800 Northeast 13th Street, Oklahoma City

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. Thomas T. Smith
Secretary-Treasurer: Dr. G. T. Alliband, 1020 Medical Arts Building, Omaha, Neb.
Time and Place: Dinner meeting third Wednesday monthly, October to May. Omaha Club

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION
Chairman: Dr. Alfred Cowan
Secretary: Dr. John W. Deichler, 301 South 21st Street, Philadelphia 3, Pa.
Time: First Thursday of each month, November through April

PHILADELPHIA LARYNGOLOGICAL SOCIETY
President: Dr. Thomas F. Furlong, Jr.
Secretary: Dr. John J. O'Keefe, 255 South 17th Street, Philadelphia 3, Pa.

PITTSBURGH ACADEMY OF MEDICINE
President: Dr. John S. Plumer
Secretary: Dr. Samuel D. Evans, 1501-2 Park Building, Pittsburgh 22, Pa.
Time and Place: 8:00 p.m., fourth Monday of each month, October through May. Pittsburgh Academy, 322 North Craig Street

PITTSBURGH OTOLOGICAL SOCIETY
President: Dr. Daniel S. DeStio
Secretary: Dr. George C. Schein, 634 Washington Road, Pittsburgh, Pa.
Time and Place: Bimonthly

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY
President: Dr. H. Frederick Thorlakson
Secretary-Treasurer: Dr. Willard F. Goff, 432 Stimson Building, 1215 Fourth Avenue, Seattle 1, Wash.
Time and Place: Third Tuesday of each month. Medical-Dental Building, Seattle

READING EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. J. Van Dyke Quereau
Secretary: Dr. Benjamin F. Souders, 424 Walnut Street, Reading, Pa.
Time and Place: Third Wednesday of each month, September to July (dinner meetings). Wyomissing Club

RICHMOND, VIRGINIA, EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Mason Smith
Secretary-Treasurer: Dr. Charles N. Romaine, 1001 West Franklin Street, Richmond 20, Va.
Time and Place: First Tuesday of January, March, May and October. Commonwealth Club

ST. LOUIS COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. Anderson C. Hilding
Secretary: Dr. James P. Tethe, 626 Medical Arts Building, Duluth, Minn.
Time and Place: 6:00 to 8:00 p.m., preceding the monthly St. Louis County Medical Society meeting. St. Mary's Hospital, Duluth

EAR, NOSE AND THROAT CLUB OF ST. LOUIS
Chairman: Dr. Millard F. Arbuckle
Secretary: Dr. Harry N. Glick, 1504 South Grand Boulevard, St. Louis 4, Mo.
Time and Place: Third Wednesday in November, January, March and May, University Club Building

ST. LOUIS OPHTHALMIC SOCIETY
President: Dr. Edmond B. Alvis
Secretary: Dr. Benjamin Milder, 539 North Grand Avenue, St. Louis 3, Mo.
Time and Place: 8:00 p.m., fourth Friday of each month, October through April, except December. Elliott Auditorium, McMillan Hospital

SAN ANTONIO SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. George H. Brunner
Secretary: Dr. Jack B. Lee, 607 New Moore Building, San Antonio, 5, Texas
Time: Second Tuesday of each month, September through May

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. W. E. Borley, 655 Sutter Street, San Francisco, Calif.
Secretary: None
Time and Place: Fourth Tuesday of each month, except July, August and December. San Francisco County Medical Society Building

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Phil Greene
Secretary: Dr. Cornelius E. Hagan, Jr., 508 Old National Bank Building, Spokane, Wash.
Time and Place: Fourth Tuesday of each month, September through May. Paulsen Medical and Dental Building

SUPERIOR CALIFORNIA EYE CLUB
President: Dr. Theodore Holstein
Secretary: Dr. John Berg, 436 Medico-Dental Bldg., Sacramento 14, Calif.
Time: Every third Thursday, eight months of the year

TOLEDO EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. N. Gardner Mathieson
Secretary: Dr. Robert D. Kiess, 1838 Parkwood, Toledo, Ohio
Time and Place: Third Tuesday of each month, November through May, Toledo Club

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY
Chairman: Dr. Joseph C. Hill
Secretary: Dr. Harold R. Sniderman, 220 Bloor Street West, Toronto 5, Ont., Canada
Time and Place: Second Monday night of November, January, February and March. Academy of Medicine, Toronto

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Ralph T. Himes
Secretary-Treasurer: Dr. F. E. Murdock, 28½ West Scribner Avenue, DuBois, Pa.
Time: Biannually. Third Thursdays of May and October

WILKES-BARRE OPHTHALMOLOGICAL SOCIETY
President: Dr. Vincent Gallizzi
Secretary: Dr. Samuel T. Buckman, 70 South Franklin Street, Wilkes-Barre, Pa.
Time and Place: Last Tuesday of each month, October through May, except December. Luzerne County Medical Society Library, 130 South Franklin Street

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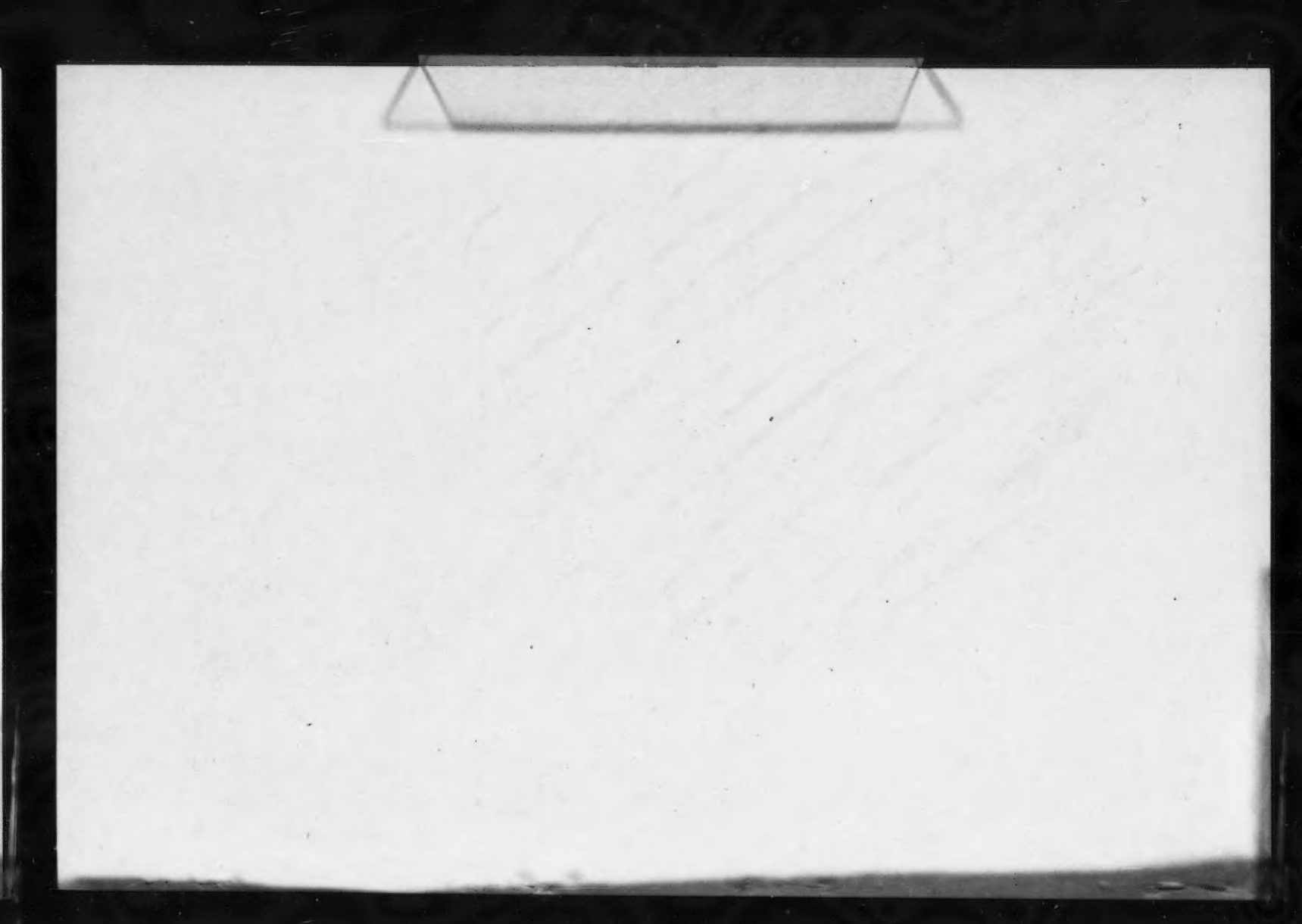

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★ BIBLIOGRAPHY

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E., E., N., & T. Mo., 26:27, 1947.
Laryngoscope, 56:556, 1946.
New Eng. J. Med., 234:468, 1946.
Annals of Allergy, 4:33, 1946.
J. A. Ph., A., (Sc. Ed.) 35:304, 1946.

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